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## ANNOUNCEMENT

RECOGNIZING the great advances in cardiovascular surgery and the increasing number of contributions by thoracic surgeons to this field, the Editorial Board and the membership of the American Association for Thoracic Surgery have with this issue officially changed the title of THE JOURNAL OF THORACIC SURGERY to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Contributions in the field of cardiovascular investigations and surgery will be acceptable only if they pertain to problems within the thorax. It is not intended that this JOURNAL publish papers dealing with the peripheral vascular system.

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# *Original Communications*

## PULMONARY ARTERY WEDGE PRESSURE IN EXPERIMENTAL MITRAL INSUFFICIENCY

*Jean Carlier, M.D.,* and Georges N. Lejeune-Ledant, M.D.,***
*Liège, Belgium*

ALTHOUGH a majority of authors[1, 2, 12, 14, 16, 24, 26] agree that, in the dog, the "mean pulmonary wedge pressure" is a faithful indication of the mean left atrial pressure, they are not of the same opinion concerning the transmission of these waves. We, therefore, have deemed it sufficiently important to investigate the transmission of the left atrial pressure to the pulmonary artery wedge pressure position in normal dogs, and in dogs with experimental mitral insufficiency and to determine, (1) whether the instantaneous changes of pressure in the left atrium are faithfully transmitted to the pulmonary artery wedge position; (2) to establish whether certain agents are capable of rendering the waves of mitral insufficiency more distinct, and (3) to study the influence of the increase of pulmonary vascular resistance on the transmission of the atrial wave of mitral insufficiency.

### MATERIALS AND METHODS

Mongrel dogs weighing between 20 and 30 Kg. were prepared under intravenous Nembutal anesthesia and intubated so that artificial respiration could be applied.

A cardiac catheter, of the type Goodale-Lubin No. 6 or No. 7, was introduced into an external jugular vein and advanced into a terminal branch of the right pulmonary artery, more frequently to the level of the inferior lobe and sometimes to the middle lobe level. It was generally wedged when the recording of the left atrial pressure began.

In some cases, a second catheter was introduced through the venous route into the pulmonary artery. In some experiments, the pulmonary artery wedge pressure was also recorded at the level of the left lower lobe.

Catheters were perfused at a slow rate (about 50 c.c. per hour) with normal saline solution containing 25 heparin units per cubic centimeter. The thorax was opened by an incision in the left fourth intercostal space. During

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*Institut de Clinique et de Policlinique Médicales (Prof. L. Brull).
**Laboratoire de Chirurgie Expérimentale (Prof. F. Albert), Université de Liège, 1, rue des Bonnes-Villes, Liège, Belgium.

the mediastinal dissection, care was taken to avoid injury of the pleura separating the two lungs. Mitral insufficiency was produced by the method of Chalnot and Benichoux,[4, 9] using a Dogliotti ring.

In experiments with an open thorax, respiration was checked except at the time of pressure recording. The left atrial pressure was measured by direct puncture with a long, fine needle connected to a No. 7 cardiac catheter.

In experiments with a closed thorax, the animal was permitted to breathe normally. Left atrial pressure was measured in 2 cases by direct transthoracic puncture with a long, fine needle. This needle was introduced into the posterior part of the third left intercostal space and directed under fluoroscopic check toward the left atrium. In other cases, the pressure was recorded by left heart catheterization via the carotid artery using a No. 6 or No. 7 cardiac catheter.

Pressures were measured with the aid of two Sanborn electromanometers, and recorded by a Sanborn Twin-Viso electrocardiograph machine using a recording speed of 5 cm. per second.

The following agents were investigated in 9 dogs with an open thorax: (1) clamping of the aorta; (2) intravenous injection of 2 to 5 units of Pitressin*; (3) intravenous injection of 0.2 mg. of epinephrine; and (4) rapid injection of normal saline (100 c.c. in 5 seconds under 1.5 Kg. pressure) using the Dos Santos apparatus,[11] via a catheter in the inferior vena cava.

In 4 dogs which had been subjected to mitral insufficiency 8 to 22 days previously, a tracheal sound connected to an anesthesia apparatus and to a water manometer allowed us to create a pressure of 20 to 30 cm. of water in the bronchi and alveoli, thus producing a compression and an increase of the pulmonary capillary bed resistance.

During this insufflation, normal saline was rapidly injected via a catheter introduced into the inferior vena cava with the aid of Dos Santos apparatus.[11]

## RESULTS

A. *Transmission of Normal Left Atrial Pressure Waves.*—

Our results in 11 dogs were as follows.

1. Except for certain deflections mostly "x" and "y," the positive waves were transmitted to the pulmonary artery wedge position with the same shape and also the same cyclic variations of amplitude (Fig. 1, Table I). The differences observed between the two positive wave tracings successively, and not simultaneously recorded, did not exceed those observed between several left auricular tracings recorded successively.

2. After opening the thorax, there was no modification of the right pulmonary artery wedge pressure, wave shape, or amplitude (Fig. 1, Table I).

3. Left pulmonary artery wedge pressure recorded on 2 dogs, after opening the thorax, revealed a damping of the wave as compared to the right pulmonary artery wedge pressure (Fig. 2).

*Pitressin = Pitone Organon.

TABLE I. NORMAL PRESSURES IN THE DOG WITH OPEN AND CLOSED THORAX (MM. Hg)

| DOG NO. | a | | | x | | | v | | | y | | | MEAN PRESSURE | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | P.A.W. | | | P.A.W. | | | P.A.W. | | | P.A.W. | | | P.A.W. | | |
| | (C.T.) | (O.T.) | L.A. | (C.T.) | (O.T.) | L.A. | (C.T.) | (O.T.) | L.A. | (C.T.) | (O.T.) | L.A. | (C.T.) | (O.T.) | L.A. |
| 1 | 6.5 | 7.5 | 7 | 3.5 | 4 | 3 | 6.5 | 7.5 | 7 | 3 5 | 3 | 1.5 | 5 5 | 5 | 4 |
| 2 | 11 | 12 | 11 | 5.5 | 6 | 4.5 | 10 | 9 | 8.5 | 6.5 | 6 | 1.5 | 7 | 6.5 | 5 |
| 3 | 11 | 10.5 | 10 | 1.5 | 1.5 | –2 | 6 | 5 | 4 | 3 5 | 2 | 1 | 5 | 4.5 | 4 |
| 4 | 9.5 | 11.5 | 11.5 | 1 | 2 | –3 | 7 | 6.5 | 7 | 1.5 | 2 | –2 | 4.5 | 4.5 | 4 |
| 5 | 5.5 | 5.5 | 5 | 3.5 | 3 | 2.5 | 8 | 7 | 5 | 3 | 3 | 2 | 5 | 4 | 3 |
| 6 | 7.5 | 7.5 | 7 | 3 | 2 | 1.5 | 7 | 6.5 | 6 | 4 | 2 | 3 | 4.5 | 3 | 3.5 |
| 7 | 10 | 8 | 7.5 | –4 | –4 | –3 | 12.5 | 12 5 | 9 | 1 | 0 | –1.5 | 5.5 | 5 | 3.5 |
| 8 | 7 | 7.5 | 6.5 | 2 | 2 | 1.5 | 5 5 | 6 | 6 | 4 | 4 | 4.5 | 3 | 3.5 | 3 |
| 9 | 6 | 8 | 8.5 | 0 | 0 | 1 | 8 | 9 | 9.5 | 0 | 0 | 1 | 2.5 | 2 | 2.5 |
| 10 | 5 | 5.5 | 6 | 2 | 2.5 | 2 | 4.5 | 4 | 4 | 2.5 | 2 5 | 1.5 | 3.5 | 3 5 | 3 |
| 11 | 7 | 6.5 | 7 | 4 | 3 | 3.5 | 7 | 6.5 | 5 | 4.5 | 4 | 4 | 6 | 5.5 | 4.5 |

L.A.—left atrial pressure; P.A.W.—pulmonary artery wedge pressure; O.T.—open thorax; C.T.—closed thorax.

TABLE II A. PRESSURES IN THE DOG WITH OPENED THORAX AFTER EXPERIMENTAL MITRAL INSUFFICIENCY (MM. Hg)

| DOG NO. | a | | x | | v | | y | | MEAN PRESSURE | |
|---|---|---|---|---|---|---|---|---|---|---|
| | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. |
| 2 | 13.5 | 15 | 7 | 7 | 14 | 15.5 | 7 | 7 | 10 | 11 |
| 3 | 24 | 23.5 | 8 | 8 | 23.5 | 24.5 | 19 | 19 | 16.5 | 15.5 |
| 4 | 11 | 8.5 | 6.5 | 6 | 17 | 18 | 5 5 | 1.5 | 11.5 | 11 |
| 5 | 10.5 | 9 | 2 | 2 | 12.5 | 12 | 7.5 | 7 | 8 | 6.5 |
| 7 | 22 | 30 | 10 | 10 | 56 | 60 | 10 | –16 | 30 | 28 |
| 8 | 5.5 | 7 | 4 | 5 | 12.5 | 14 | 4 | 0 | 8 | 7 |
| 9 | 13 5 | 15 | 4 | 5 | 13 | 15 | 5.5 | 0 | 7.5 | 7 |
| 10 | 8.5 | 8 | 4.5 | 4 | 11.5 | 13 | 5 | 2 | 8 | 7 |
| 11 | – | – | 18 | 20 | 56 | 52 | 19 | –8 | 28 | 26.5 |

TABLE II B

| DOG NO. | a | | x | | v | | y | | MEAN PRESSURE | |
|---|---|---|---|---|---|---|---|---|---|---|
| | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.P.A.W. | R.P.A W. | L.P.A.W. |
| 4 | 11 | 9 | 6.5 | 7.5 | 17 | 12.5 | 5.5 | 6 | 11.5 | 10.5 |
| 8 | 5.5 | 5.5 | 4 | 4.5 | 12.5 | 8 | 4 | 5 | 7.5 | 7 |

R.P.A.W.—right pulmonary artery wedge pressure; L.A.—left atrial pressure; L.P.A.W.—left pulmonary artery wedge pressure.

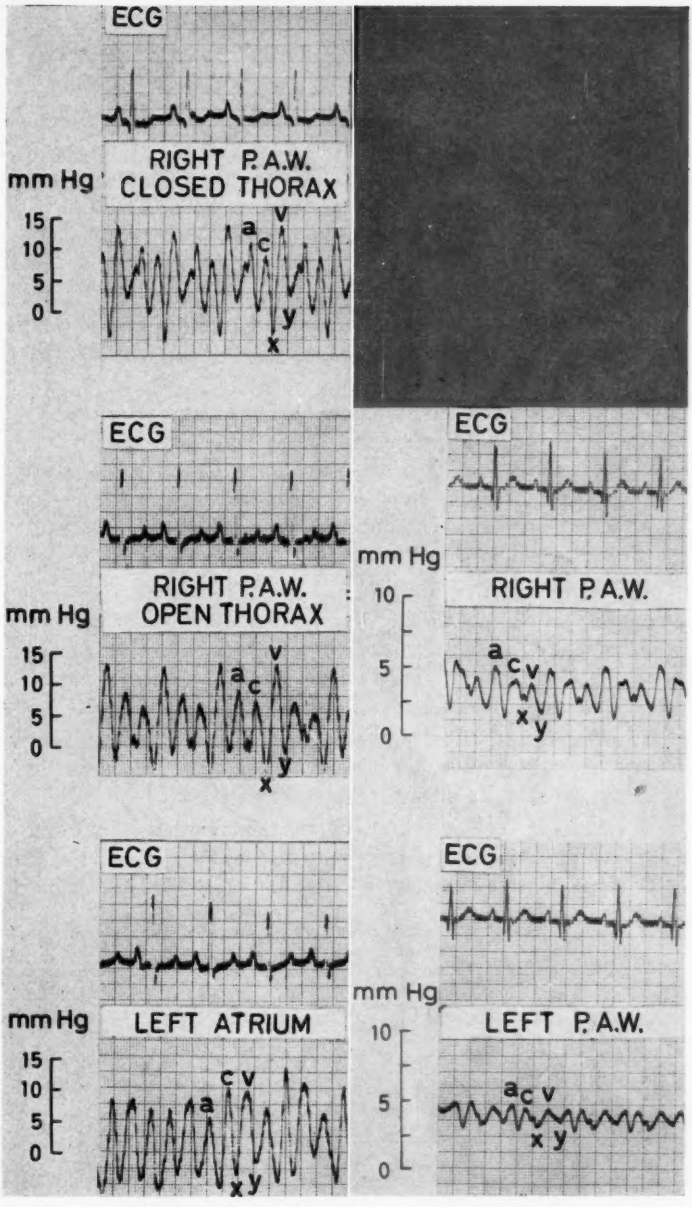


Fig. 1. Fig. 2.

Fig. 1.—(Dog 7.) Right P.A.W.—right pulmonary artery wedge. On the left atrial tracing, three waves "*a*," "*c*," and "*v*" can be seen. They are transmitted to the right pulmonary artery wedge position with equal amplitude. The right pulmonary artery wedge tracing with open and closed thorax is the same.

Fig. 2.—(Dog 10.) Right P.A.W.—right pulmonary artery wedge; Left P.A.W.—left pulmonary artery wedge. The pulmonary artery wedge tracing recorded on the side of the thoracotomy (left side) enables us to recognize the three atrial waves, but they are less ample than on the opposite side.

B. *Transmission of the Left Atrial Pressure Wave in Mitral Insufficiency.*—

1. *Experiments in 9 dogs with an open thorax:*

a. In 7 dogs, a typical mitral insufficiency wave was recorded in atrial tracings. This wave was found on the right pulmonary artery wedge pressure tracing. It was also identical both in form and amplitude with the atrial wave (Fig. 3, Table II A).

b. In 2 dogs showing a definite moderate mitral insufficiency verified by finger palpation and at autopsy, the right pulmonary artery wedge tracing did not show a typical regurgitation "v" wave. The left atrial curve, furthermore, did not reveal a characteristic mitral insufficiency wave and its shape was the same as that of the pulmonary artery wedge tracing.

c. In 2 dogs, we recorded the pulmonary artery wedge pressure in both lungs (Table II B). The left mean pulmonary artery wedge pressure was the same as in the right lung, but the cyclic variations of the left lung reading were "damped." The left wave was still visible although less apparent and of lesser amplitude. Its peak was retarded as compared with the right pulmonary artery wedge pressure tracing by a time interval of 0.05 to 0.06 second.

2. *Dogs with a closed thorax:* Twelve dogs underwent an experimental mitral insufficiency. Six of these dogs developed a severe mitral insufficiency and died of acute pulmonary edema within a week. A 1 to 3 week follow-up study of the remaining operated dogs showed the following results.

a. The regurgitation "v" wave on the left atrial tracing was seen also on the right pulmonary artery wedge pressure curve and was of about the same amplitude (Fig. 4, Table III A).

b. In spite of the resorption of the pneumothorax and the absence of liquid in the left pleura on 2 of the dogs examined 15 days and 3 weeks postoperatively, a damping of the left pulmonary artery

TABLE III A. PRESSURES IN THE DOG WITH CLOSED THORAX AFTER EXPERIMENTAL MITRAL INSUFFICIENCY (MM. Hg)

| DOG | a | | x | | v | | y | | MEAN PRESSURE | |
|---|---|---|---|---|---|---|---|---|---|---|
| NO. | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. | R.P.A.W. | L.A. |
| 1 | 10 | 10 | 5 | 4 | 10 | 10.5 | 6.5 | 3 | 6.5 | 5 |
| 2 | 6 | 5 | 5 | 4 | 15 | 16 | 6 | 3 | 7.5 | 6 |
| 3 | 7 | 7.5 | 5 | 4 | 17 | 15 | 5 | 3 | 7 | 6 |
| 4 | 5.5 | 4 | 2.5 | 1.5 | 8 | 7.5 | 2.5 | 1 | 5 | 3.5 |
| 5 | 8.5 | 9 | –1 | –1 | 11.5 | 11.5 | 3 | 0 | 5 | 4 |
| 6 | 13 | 13 | 6 | 5 | 11.5 | 12 | 5 | 3 | 7.5 | 7 |

TABLE III B

| DOG | a | | x | | v | | y | | MEAN PRESSURE | |
|---|---|---|---|---|---|---|---|---|---|---|
| NO. | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.P.A.W. | R.P.A.W. | L.A. | R.P.A.W. | L.P.A.W. |
| 3 | 7 | 6 | 5 | 5.5 | 17 | 10 | 5 | 6 | 7 | 6.5 |
| 4 | 5.5 | 5 | 2.5 | 4 | 8 | 5.5 | 2.5 | 4.5 | 5 | 5 |

R.P.A.W.—right pulmonary artery wedge pressure; L.A.—left atrial pressure; L.R.A.W.—left pulmonary artery wedge pressure.

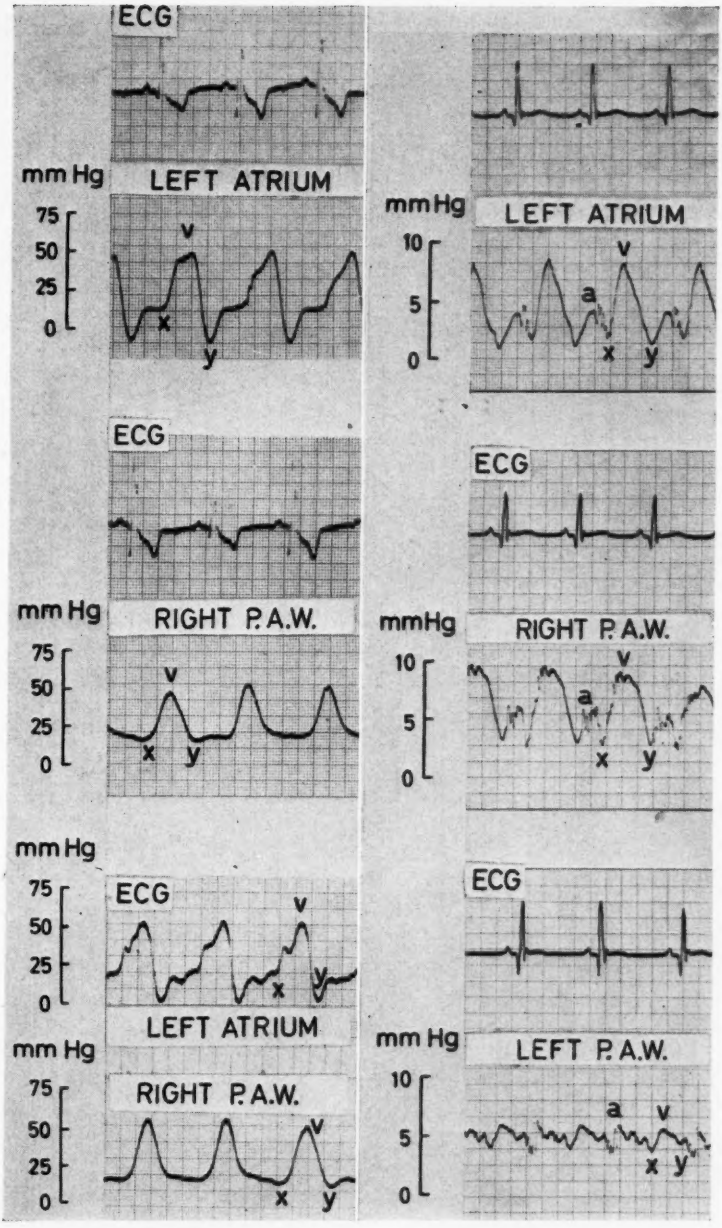


Fig. 3.

Fig. 4.

Fig. 3.—(Dog 11.) Right P.A.W.—right pulmonary artery wedge. On the left atrial tracing, there is a typical "v" wave of mitral regurgitation. This wave is transmitted to the right pulmonary artery wedge position with the same amplitude. The difference between the two tracings is due to an absence of transmission of the left atrial "y" wave below the "x" wave.

Fig. 4.—(Dog 4.) Right P.A.W.—right pulmonary artery wedge; Left P.A.W.—left pulmonary artery wedge. The left atrial "v" wave of mitral insufficiency is transmitted to the right pulmonary artery wedge position with the same amplitude. On the left side, there is an important damping of the atrial waves, mainly the "v" wave, the apex of which is delayed with respect to that of the tracing recorded on the right side. The mean pressures of the left and right pulmonary artery wedge positions are identical and slightly greater than that of the left atrium.

TABLE IV

*Influence of the Clamping of the Aorta (mm. Hg)*

| DOG NO. | a BEFORE R.P. A.W. | a BEFORE L.A. | a DURING R.P. A.W. | a DURING L.A. | x BEFORE R.P. A.W. | x BEFORE L.A. | x DURING R.P. A.W. | x DURING L.A. | v BEFORE R.P. A.W. | v BEFORE L.A. | v DURING R.P. A.W. | v DURING L.A. | y BEFORE R.P. A.W. | y BEFORE L.A. | y DURING R.P. A.W. | y DURING L.A. | MEAN PRESSURE BEFORE R.P. A.W. | MEAN PRESSURE BEFORE L.A. | MEAN PRESSURE DURING R.P. A.W. | MEAN PRESSURE DURING L.A. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 18 | 16.5 | 23 | 22 | 8 | 8 | 20 | 18 | 34 | 33 | 62 | 61 | 10 | 2 | 20 | 8 | 25 | 23 | 43 | 40.5 |
| 2 | 20 | 21 | 25 | 25.5 | 11 | 8 | 22 | 21 | 30 | 28.5 | 54 | 56 | 11 | 7 | 21 | 0 | 14 | 12 | 29.5 | 26.6 |
| 3 | 24 | 23.5 | 33 | 38.5 | 18 | 18 | 30 | 33 | 23.5 | 23.5 | 57 | 57 | 3 | 1 | 27 | 13 | 16.5 | 15.5 | 34 | 31 |
| 4 | 26 | 20 | 25.5 | 23 | 21 | 17 | 30 | 29.5 | 46 | 46 | 71 | 71 | 23 | 0 | 30 | 4 | 39 | 37 | 49 | 46 |
| 5 | 38 | 34 | 43 | 42 | 29 | 30 | 37 | 35 | 57 | 58 | 107 | 109 | 31 | 21 | 36 | 25 | 40 | 36 | 68 | 64 |

*Influence of Intravenous Injection of Pitressin (mm. Hg)*

| DOG NO. | DOSAGE | a BEFORE R.P. A.W. | a BEFORE L.A. | a AFTER R.P. A.W. | a AFTER L.A. | x BEFORE R.P. A.W. | x BEFORE L.A. | x AFTER R.P. A.W. | x AFTER L.A. | v BEFORE R.P. A.W. | v BEFORE L.A. | v AFTER R.P. A.W. | v AFTER L.A. | y BEFORE R.P. A.W. | y BEFORE L.A. | y AFTER R.P. A.W. | y AFTER L.A. | MEAN PRESSURE BEFORE R.P. A.W. | MEAN PRESSURE BEFORE L.A. | MEAN PRESSURE AFTER R.P. A.W. | MEAN PRESSURE AFTER L.A. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2U | 14 | 12.5 | 21 | 23 | 6 | 6 | 12 | 10 | 21 | 22 | 48 | 47 | 7.5 | 0 | 12 | 3 | 14 | 13 | 37 | 35 |
| 2 | 2U | 13.5 | 15 | 20 | 21 | 7 | 7 | 11 | 8 | 14 | 15.5 | 30 | 28.5 | 7 | 7 | 11 | 7 | 10 | 11 | 21 | 21 |
| 3 | 4U | 24 | 23.5 | 41 | 43 | 18 | 18 | 34 | 32 | 23.5 | 23.5 | 89 | 86 | 19 | 19 | 34 | 0 | 16.5 | 15.5 | 58 | 56 |
| 4 | 4U | 11 | 8.5 | 39 | 41 | 6.5 | 6 | 32 | 34 | 17 | 18 | 83 | 85 | 5.5 | 1.5 | 25 | 0 | 11.5 | 11 | 68 | 65 |
| 5 | 5U | 10.5 | 9 | 38 | 34 | 2 | 2 | 29 | 30 | 12.5 | 12 | 57 | 58 | 7.5 | 7 | 31 | 21 | 7 | 6.3 | 40 | 36 |
| 8 | 4U | 5.5 | 7 | 21 | 19 | 4.5 | 5 | 16.5 | 10 | 12.5 | 14 | 125 | 125 | 4.5 | 1 | 14.5 | 0 | 8.5 | 7 | 72 | 68 |

*Influence of Intravenous Injection of 0.2 mg. of Epinephrine (mm. Hg)*

| DOG NO. | a BEFORE R.P. A.W. | a BEFORE L.A. | a AFTER R.P. A.W. | a AFTER L.A. | x BEFORE R.P. A.W. | x BEFORE L.A. | x AFTER R.P. A.W. | x AFTER L.A. | v BEFORE R.P. A.W. | v BEFORE L.A. | v AFTER R.P. A.W. | v AFTER L.A. | y BEFORE R.P. A.W. | y BEFORE L.A. | y AFTER R.P. A.W. | y AFTER L.A. | MEAN PRESSURE BEFORE R.P. A.W. | MEAN PRESSURE BEFORE L.A. | MEAN PRESSURE AFTER R.P. A.W. | MEAN PRESSURE AFTER L.A. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 7 | – | – | – | – | – | – | – | – | 72 | 80 | 126 | 121 | 22 | 25 | 25 | 25 | 48 | 52 | 78 | 75 |
| 9 | 13.5 | 15 | 19 | 20 | 4 | 5 | 6 | 7 | 13 | 15 | 27 | 26 | 5.5 | 0 | 9 | 2 | 7.5 | 7 | 14 | 13 |
| 10 | 8.5 | 8 | 13 | 12 | 4.5 | 4 | 7 | 5 | 11.5 | 13 | 29 | 27.5 | 5 | 2 | 9 | 7 | 8 | 7 | 17 | 14 |

*Influence of the Rapid Injection of 100 c.c. of Normal Saline (mm. Hg)*

| DOG NO. | a BEFORE R.P. A.W. | a BEFORE L.A. | a DURING R.P. A.W. | a DURING L.A. | x BEFORE R.P. A.W. | x BEFORE L.A. | x DURING R.P. A.W. | x DURING L.A. | v BEFORE R.P. A.W. | v BEFORE L.A. | v DURING R.P. A.W. | v DURING L.A. | y BEFORE R.P. A.W. | y BEFORE L.A. | y DURING R.P. A.W. | y DURING L.A. | AFTER THE INJECTION v R.P. A.W. | AFTER THE INJECTION v L.A. | AFTER THE INJECTION y R.P. A.W. | AFTER THE INJECTION y L.A. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 5 (B) | 8.5 | 8 | 13 | 12 | 4.5 | 4 | 7 | 6.5 | 6 | 5 | 14 | 12 | 3.5 | 3 | 7 | 5 | 9.5 | 8.5 | 5.5 | 4 |
| 8 (B) | – | – | – | – | – | – | – | – | 35.5 | 36 | 62 | 57 | 8.5 | 7 | 15 | 12 | 34 | 35 | 10 | 9 |
| 9 | 13.5 | 15 | 19 | 20 | 4 | 5 | 6 | 7 | 13 | 15 | 29.5 | 31 | 5.5 | 0 | 8 | 3 | 16 | 15.5 | 6.5 | 1 |
| 10 | – | – | – | – | – | – | – | – | 11.5 | 13 | 23 | 24 | 5 | 2 | 9 | 6 | 14 | 14.5 | 6.5 | 4 |

R.P.A.W.—right pulmonary artery wedge; L.A.—left atrium.

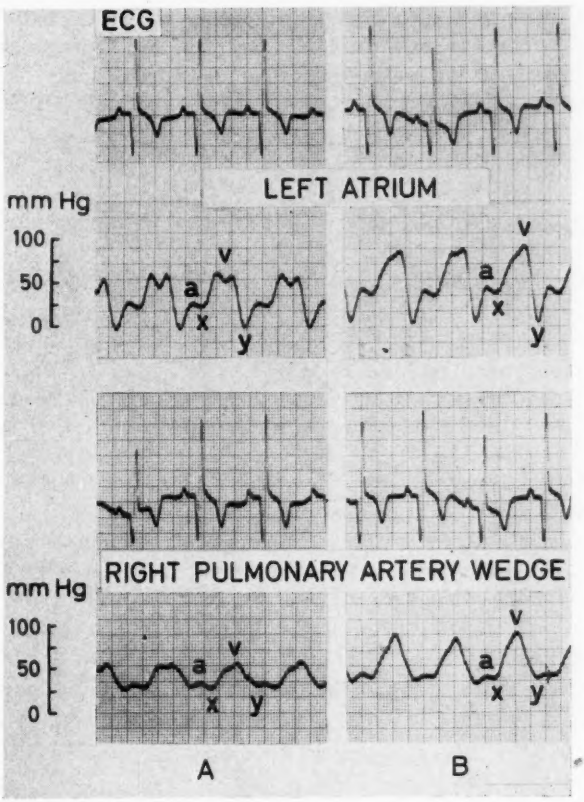


Fig. 5.—Influence of aortic clamping. *A*, Before; *B*, after. The increase of pressure of the regurgitation "v" wave is equal in the two positions.

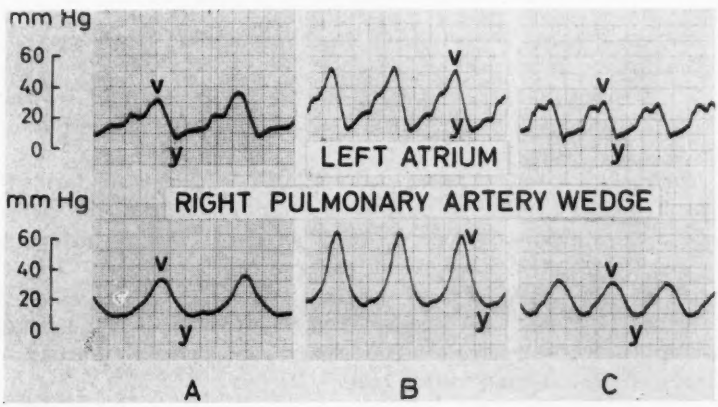


Fig. 6.—The influence of a rapid intravenous injection of 100 c.c. of physiologic liquid. *A*, Before. *B*, Immediately after. *C*, 3 minutes after.

R.P.A.W.—right pulmonary artery wedge; L.A.—left atrium.

wedge pressure, involving principally the regurgitation "v" wave, was found (Fig. 4, Table III B). The mean pressure was exactly the same as the pressure recorded for the right lung.

C. *Action of Various Agents on the Pressure Curves of the Left Atrium and the Pulmonary Artery Wedge Pressure in Experimental Mitral Insufficiency* (see Table IV and Figs. 5 and 6).—

1. For the first three agents (clamping of the aorta, intravenous injection of 2 to 5 units of Pitressin, intravenous injection of 0.2 mg. of epinephrine) the following results were noted.

a. The increase of the mean left atrial pressure observed was transmitted to the pulmonary artery wedge, the pressure in the latter being mostly a few millimeters of mercury greater than the left atrial pressure (Table IV).

b. These agents produced an increase of the atrial regurgitation "v" wave that was much more important than that of the "a" wave (Table IV, Fig. 5).

c. The pulmonary artery wedge tracing faithfully followed the modifications observed at the positive waves, mainly at the "v" wave regurgitation. Whereas, on the initial tracing, the negative "x" wave was generally well transmitted, the pulmonary artery wedge "y" wave often did not go below the "x" level (Table IV, Fig. 5).

2. The rapid injection of normal saline solution produced a definite and transitory increase of the "v" wave, more apparent than for the "a" wave, the corresponding pressures at "x" and "y" being only slightly increased (Table IV, Fig. 6).

D. *Influence of the Increase of Pulmonary Vascular Resistance on the Transmission of the Left Atrial Waves.*—

1. During the insufflation of the lungs under pressure, a damping of the left atrial and pulmonary artery wedge tracings was observed (Fig. 7, *A* and *B*).

2. One also noticed a damping of the pulmonary artery wedge "v" wave as compared to the wave recorded in the left atrium.

3. The transmission time of the atrial "v" wave (measured from the apex of the wave) to the pulmonary artery wedge position was increased (Fig. 7, *A* and *B*).

4. The rapid injection of normal saline solution intravenously during the insufflation of the lung under pressure caused, on the one hand, an increase of the "v" waves which was more apparent on the pulmonary artery wedge tracing and, on the other hand, a shortening of the transmission delay of the left atrial "v" wave (Fig. 7, *C*).

5. Immediately after the end of insufflation of the lungs, a transitory increase of the "v" wave was observed both on the pulmonary artery wedge tracing and on the left atrial tracing (Fig. 7, *D*).

6. With respect to the pulmonary artery tracing, during the insufflation, a damping of the pulmonary artery wedge "v" wave was observed, as well as a

delay in the recording of its apex with respect to the apex of the pulmonary arterial systolic wave.

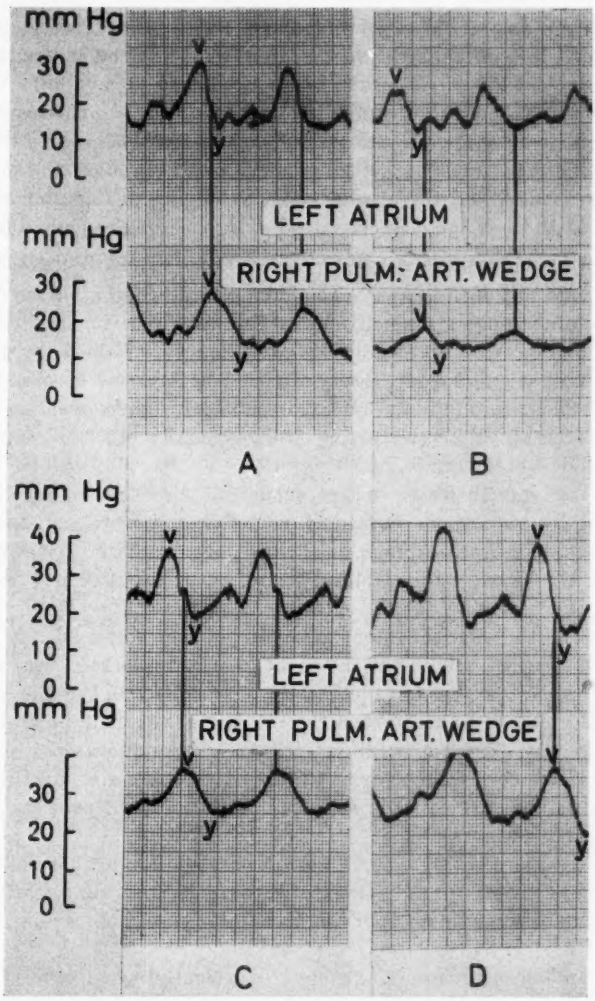


Fig. 7.—Influence of rapid intravenous injection of 100 c.c. of normal saline during the lung insufflation.

*A*, Before the lung insufflation.

*B*, During the insufflation under a pressure of 30 cm. water.

*C*, Immediately after the rapid intravenous injection of 100 c.c. of normal saline during the lung insufflation.

*D*, Immediately after the lung insufflation.

On the *B* tracing, a damping and a supplementary transmission delay of the apex of the P.A.W. "v" wave is observed, with respect to that of the left atrium. The rapid injection of normal saline almost restores the P.A.W. "v" wave to its initial amplitude and suppresses the supplementary delay of transmission of the left atrial "v" wave to the P.A.W. position.

COMMENTS

A. *Transmission of the Normal Left Atrial Pressure.*—

In our experiments, the thorax was opened by a left intercostal incision. The catheter tip was at the right lung level. The thin pleura separating the two lungs was left untouched and therefore a right-sided pneumothorax was avoided.

This condition must be fulfilled in order to have a good transmission of the left atrial waves toward the pulmonary artery wedge position. In two "open-thorax" experiments, the pulmonary artery wedge pressure in both lungs was recorded, and there was decisive evidence of damping on the pressure curve of the left lung as compared with that of the right lung.

B. *Transmission of the Atrial Pressure Curve in Mitral Insufficiency.*—

In the case of "open-thorax" dogs having undergone a mitral insufficiency by a left thoracotomy approach, under our own experimental conditions, a good transmission of the atrial waves and, mainly, of the regurgitation "v" wave to the right pulmonary artery wedge position was observed. In some dogs, which in spite of proved mitral insufficiency, there was no typical "v" wave on the right pulmonary artery wedge position, the "v" wave was also absent in the left atrial curve.

In similar human cases where moderately severe mitral insufficiency was verified by palpation, regurgitation "v" waves were not discernible on left atrial curves. These human cases have been reported by Wynn and his collaborators,[28] Venner and Holling[25] and, also, by Ankeney and his colleagues.[3]

As regards the dog, the effect of a left pneumothorax on left atrial wave transmissions can be seen. On the right side, where there was no pneumothorax, all atrial waves were correctly transmitted. On the left side, a damping effect could be seen when the recording was made in the pulmonary artery wedge position. A midline incision of the thorax, as practiced by Ankeney and his co-workers,[2] or a rupture of the pleura separating the lungs during a left-sided surgical approach, can be the cause of poor transmission of regurgitation "v" waves.

In "closed-thorax" dogs, our findings were similar to those of Haddy and his co-workers[14] who found a good transmission of regurgitation waves 2 weeks later in operated dogs. In this particular case, the wave amplitude was of 40 mm. Hg.

For even lower pressures, we have experienced good wave transmission in our results. Coelho and associates[10] were not able to put into evidence any typical pulmonary artery wedge pressure on "closed-thorax" dogs when the mitral valves were experimentally rendered insufficient.

Even without any liquid reaction of the pleura, the effect of a left pneumothorax on the transmission of atrial waves continued, even when the pneumothorax was completely resorbed. In fact, 15 days to 3 weeks after operation, a damping of the pulmonary artery wedge pressure curve at the level of the inferior left lobe has still been observed in 2 dogs.

C. *Action of Various Agents on the Pressure Curves of the Left Atrium and the Pulmonary Artery Wedge Pressure in Experimental Mitral Insufficiency.*—

In the dog with the "closed-thorax," Vuylsteek[26] noted that the increase of mean left atrial pressure produced by the obliteration of the aorta, or the injection of epinephrine or Pitressin, was transmitted to the pulmonary artery wedge position. Our experiments confirmed Vuylsteek's findings on the dog with the open thorax and also showed that the increase of the left atrial "a" and "v" waves are found practically to be equal on the pulmonary artery wedge tracings.

These three agents, as well as the rapid intravenous injection of normal saline solution produced an increase of the regurgitation "v" wave which was more important than that of the wave "a"; it was only temporary in the case of injection of normal saline solution.

After the latter test, left atrial and pulmonary artery wedge diastolic pressures usually did not return to the initial level. Ankeney,[1, 2] and Henry[17] and Ferguson[13] and their associates observed the same increase of the left atrial diastolic pressure after injection of liquid of approximately the same volume as that used in our own experiment. This might also be caused by the increase of the blood volume.

D. *Influence of the Increase of Pulmonary Vascular Resistance on the Transmission of the Left Atrial Waves in Experimental Mitral Insufficiency.*—

1. The increase of pulmonary vascular resistances in human mitral disease is a well-known phenomenon. On the contrary, in the dog subjected to a mitral stenosis, Haddy and collaborators[15] proved that after 10 months there was no increase of these resistances. Owing to the fact that the time required for the setting of these increased resistances in the dog is certainly longer than 10 months, we preferred to use a simple and rapid technique which consists of insufflation of the lungs of the experimental animal under pressure. Its mechanism is certainly not the same as in human mitral disease but the place where the increase of the resistance acts is of little importance for our study. The decrease of the pulmonary vascular network caliber produced by insufflation under pressure is without doubt, if one refers to the fine angiopneumographies of Rimini.[20]

2. Our observations confirm the damping of the pulmonary artery wedge tracing observed in man by Shaffer and Silber[21] in the case of mitral disease with increase of pulmonary arteriolar resistances, as well as by Björk,[6] Lee,[19] during Valsalva's test, and Werkö[27] and their collaborators during positive respiration.

3. The delay in the transmission of the left atrial regurgitation wave observed by comparing the pulmonary artery wedge "v" wave to the left atrial "v" wave or to the pulmonary artery tracing showed a shortcoming during the determination of mitral insufficiency in man on a pulmonary artery wedge tracing.

It should be noted that this transmission delay, associated with a damping of the "v" wave, may result in the lowering of the apex of the pulmonary artery wedge "v" wave after the closing wave of the pulmonary artery. Its precession in respect of the latter was admitted by Lagerlöf and Werkö,[18] and Soulie and co-workers[22] as being a criterion of mitral insufficiency.

More recently, some authors, among whom Soulie[23] and Blum[7] and their associates have considered this criterion (precession of the pulmonary artery wedge "v" wave in respect to the closing wave of pulmonary artery) as being unreliable. Soulie admits, however, that in the presence of regular sinus rhythm, and in determined conditions, it may be a useful adjuvant in diagnosis. Soulie and collaborators[23] have also argued that the "pulmonary artery wedge" tracing could, in the case of mitral insufficiency, have a different morphology depending on the territory where it is recorded and on the tension applied to the extremity of the catheter.

The modifications observed by these authors may be explained in the following manner: (1) the variable anatomic alterations in one or another place of the lung also suggest various vascular resistances, (2) the relations between the vascular walls and the catheter orifice with an exaggerated tension may obliterate, at least partly, the catheter orifice (these two factors are an obstacle to the transmission on the regurgitation wave and may offset its apex with respect to the pulmonary artery closing wave), and (3) the rapid injection of normal saline solution, creating a sudden distention of the pulmonary vascular bed, practically restores the regurgitation wave "v" to its initial amplitude and suppresses the additional delay of the transmission of the left atrial "v" wave created by the insufflation of the lungs.

However, we cannot consider this technique as being free from danger as far as man is concerned. Yet it is possible that immediately after a Valsalva test, which creates a sudden and transitory flow into the lungs, the recording of the pulmonary artery wedge curve brings about modifications similar to those observed during the rapid injection of normal saline solution. Providing the cardiac output can be sufficiently increased, an effort test could achieve the same result.

## CONCLUSIONS

1. In normal dogs having undergone a left thoracotomy, there was a good transmission of the positive left atrial waves toward the right pulmonary artery position. The left position tracing was damped. The opening of the thorax modified neither the form nor the amplitude of the right pulmonary artery wedge pressure tracing.

2. In the dog submitted to an experimental mitral insufficiency by a left thoracotomy approach, the left atrial regurgitation wave was correctly transmitted toward the right pulmonary artery wedge position. In dogs in which no regurgitation wave was discernible on the right pulmonary artery wedge position tracing, the wave was also absent on the left atrial recording. A

pneumothorax gave rise to a damping of the left pulmonary artery wedge pressure curve as compared to the right, and a retardation of the peak of the regurgitation wave.

3. In "closed-thorax" dogs having undergone a mitral insufficiency and breathing normally, examined 1 to 3 weeks after, a good transmission of the left atrial regurgitation wave toward the right pulmonary artery wedge position was found as experienced in "open-thorax" dogs under artificial respiration. A damping of the left pulmonary artery wedge pressure tracing still occurred, in spite of total pulmonary re-expansion and absence of liquid in the pleural cavity.

4. Influence of mechanical or pharmacodynamic agents: (a) In dogs subjected to mitral insufficiency, the obliteration of the aorta and intravenous injection of Pitressin or epinephrine produced an increase of the mean left atrial pressure as well as the amplitude of the regurgitation wave. (b) These modifications were faithfully transmitted to the right pulmonary artery wedge position. (c) A rapid intravenous injection of normal saline determined a definite but transitory increase of the regurgitation wave in equal proportions, both on the left atrial tracing and on the pulmonary artery wedge tracing.

5. Influence of the increase of pulmonary vascular resistance: (a) The increase of pulmonary vascular resistances created by the insufflation of the lungs under pressure produced a damping and an additional delay in the transmission of the mitral insufficiency wave to the pulmonary artery wedge position. (b) During the insufflation of the lungs under pressure, the rapid intravenous injection of normal saline restored the pulmonary artery wedge regurgitation wave practically to its initial amplitude and cancelled the additional delay in the transmission to the pulmonary artery wedge position.

## SUMMARY

In dogs with open and closed thoraces, there was a good transmission of the mean left atrial pressure to the pulmonary artery wedge position.

The normal and pathologic (mitral insufficiency) positive waves were faithfully transmitted only to the side opposite the thoracotomy. On the operated side, there was a damping of the pulmonary artery wedge tracing which persisted for at least 3 weeks after the thoracotomy.

The mitral regurgitation wave could be considerably increased under the influence of various mechanical or pharmacodynamic agents. These modifications were faithfully transmitted to the pulmonary artery wedge position. The increase of pulmonary vascular resistance created a damping and a delay in the transmission of the mitral regurgitation wave on the pulmonary artery wedge tracing. These modifications temporarily disappeared through a rapid intravenous injection test of normal saline solution.

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# PROLONGED VENTRICULAR FIBRILLATION

## An Experimental Study in the Closed Chest Animal Utilizing a Pump-Oxygenator

*John W. Bell, M.D., and Francesco F. Beretta, M.D.,* Seattle, Wash.*

### INTRODUCTION

Prior to the development of the pump-oxygenator and realization of circulatory substitution, ventricular fibrillation was well known as a fatal and irreversible arrhythmia. Without support of the coronary and cerebral circulation, experience proved that the fibrillating heart, after several minutes, could seldom be restored to a satisfactory rhythm either by chemical or electrical means.[1, 2] Since the advent of intracardiac surgery, evidence is accumulating that ventricular fibrillation is a relatively innocuous arrhythmia when it occurs during pump-oxygenator support.[3]

Our interest in the deliberate use of ventricular fibrillation in the intact animal resulted from a search for a satisfactory method of producing cardiogenic shock. This arrhythmia appeared to satisfy the criteria of an experimental state which was reversible, easily induced, and during which the animal was totally dependent upon extracorporeal support.

Our purpose is to report satisfactory prolonged substitution for the fibrillating canine heart with the bubble oxygenator. These experiments were done with peripheral cannulation of the femoral vessels in the normothermic, closed chest animal. Successful electrical defibrillation of the heart following 2 to 3 hours of deliberate fibrillation, is in accord with Wiggers' early demonstration that a well-oxygenated myocardia is the vital requirement for restoration of sinus rhythm.[4]

### METHODS

Ten healthy mongrel dogs varying in weight from 12 to 34 Kg. were anesthetized with intravenous sodium pentobarbital, intubated, and mechanically respirated.

From the Department of Surgery, Veterans Administration Hospital, and University of Washington School of Medicine, Seattle, Wash.

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*Formerly Senior Resident in Surgery, Veterans Administration Hospital, Seattle, Washington.

Connection to the oxygenator was accomplished by peripheral cannulation. The largest catheter possible (usually 20 Fr.) was introduced through the left femoral vein into the inferior vena cava to the level of the diaphragm. For arterial input the right femoral artery was cannulated in a retrograde manner.

Systemic arterial pressure was measured by catheterization of the left femoral artery and connection to a Statham P23D pressure transducer. Recordings were made on a Sanborn Twin-Viso and monitored on the Viso-Scope. Central venous pressure was measured with a water manometer by cannulation of the right jugular vein. The electrocardiogram (Lead II) was recorded on a Sanborn Cardiette and monitored as necessary on the Viso-Scope. The animal was given intravenous heparin, 1 mg. per kilogram. The blood in the oxygenator system was heparinized at 3 mg. per 100 ml. Animal temperature and temperature of the blood in the debubbling chamber were measured with thermistor probes and a Yellow Springs telethermometer.

Ventricular fibrillation was induced using external chest electrodes. The Morris apparatus, capable of voltage up to 220 and impulse duration of 0.5, 1.0 sec., has been used. Evidence of ventricular fibrillation was noted by absence of the apical impulse, the characteristic fibrillatory pattern on the electrocardiogram and the immediate drop of arterial pressure to a static level of 5 to 10 mm. Hg. Following the onset of fibrillation, extracardiac substitution was begun, usually within 10 to 30 seconds. Venous pH was drawn at 15-minute intervals. Serum potassium was determined prior to defibrillation in occasional experiments.

After varying periods of fibrillation, the heart was defibrillated through the intact chest, using large external electrodes 8.5 cm. in diameter. For this purpose the Electrodyne defibrillator,* capable of 750 volts at 0.15 sec., has been successful.

## RESULTS AND OBSERVATIONS

*Technique of Fibrillation and Defibrillation.*—The electrical factors necessary for the induction of ventricular fibrillation in the intact animal were established largely by trial. It may be seen from Table I that there is a rough correlation of animal weight to fibrillation voltage. Dogs, from 10 to 20 Kg., usually required 110 to 135 volts. The larger dogs, from 20 to 30 Kg., required 135 to 160 volts at 0.5 sec. duration. An impulse duration of less than 0.5 sec. was not reliable for fibrillation and a shock of 1.0 sec. was no more effective than 0.5 sec. Although body weight altered fibrillation voltage and time requirement, equally important in the intact animal was the amount of muscle and fat on the chest wall and the position and contact of the external electrodes. On the exposed canine heart, Mackay and associates[5] found that currents of low value and long duration were necessary to produce fibrillation. Early work by Hooker, Kouwenhoven, and Langworthy[6] demonstrated the ease with which ventricular fibrillation may be established in the canine heart by relatively low

*Electrodyne Co., Inc., Norwood, Mass.

currents. They found that one milliampere of current, applied directly to the ventricular musculature, was sufficient to cause fibrillation, and the extreme apex was as sensitive as any other point in the ventricles.

For successful defibrillation in the intact animal, due to the spread of current over the tissues on either side of the thorax, the countershock must be increased three to four times the value required for fibrillation. Kouwenhoven and co-workers[7] state that 4 or 5 amperes is necessary, while Guyton and

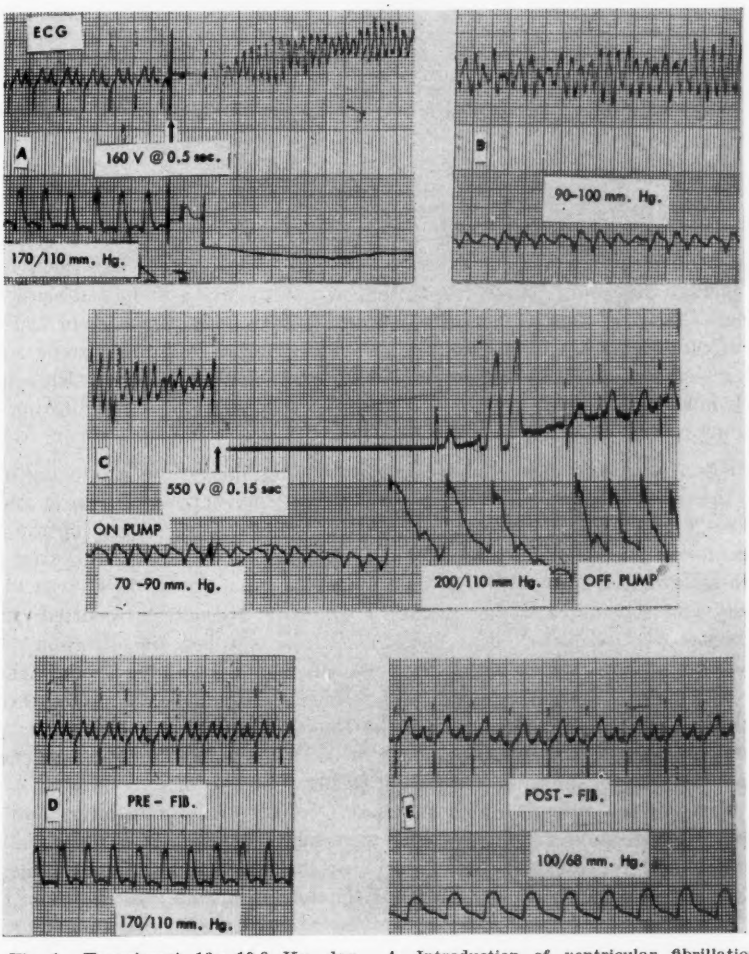


Fig. 1.—Experiment 13; 19.8 Kg. dog. *A*, Introduction of ventricular fibrillation in closed chest animal.

*B*, After 2 hours of ventricular fibrillation, electrocardiogram shows fine wave with good amplitude. Mean arterial pressure of 90 mm. Hg with perfusion of 65 to 70 ml./Kg.

*C*, Defibrillation after 2½ hours of total body perfusion. Prompt resumption of sinus rhythm and exaggerated systemic arterial pressure with hypervolemia.

*D*, Prefibrillatory electrocardiogram and arterial pressure pattern (compare to *E*) 4 to 5 minutes after defibrillation.

*E*, Initial hypervolemia restored to normovolemia.

Satterfield[8] believe approximately 20 amperes to be satisfactory. They found, as have other investigators, that the important factor in defibrillation is the density of current flow through the heart. They showed that large external electrodes caused the greatest density of current through the heart for a given voltage.

TABLE I. ELECTRICAL FACTORS IN CLOSED CHEST CARDIAC FIBRILLATION AND DEFIBRILLATION

| EXPERIMENT | WEIGHT (KG.) | SUBFIBRILLATION | FIBRILLATION | DEFIBRILLATION |
|---|---|---|---|---|
| 6 | 17 | | 0.5 sec. 110v. | 0.15 sec. 250v. |
| 7 | 34 | | 0.5 sec. 135v. | 0.15 sec. 550v. |
| 8 | 14 | | 0.5 sec. 110v. | 0.15 sec. 350v. |
| 9 | 22 | 0.5 sec. 110v. | 0.5 sec. 135v. | 0.15 sec. 650v. |
| 10 | 19 | 1.0 sec. 85v. | 0.5 sec. 135v. | 0.15 sec. 550v. |
| 11 | 16.2 | 0.5 sec. 135v. | 0.5 sec. 160v. | 0.15 sec. 550v. |
| 12 | 24 | | 0.5 sec. 160v. | 0.15 sec. 550v. |
| 13 | 19.8 | | 0.5 sec. 160v. | 0.15 sec. 550v. |
| 14 | 14.5 | | 0.5 sec. 160v. | 0.15 sec. 550v. |
| 29 | 20 | | 0.5 sec. 135v. | 0.15 sec. 550v. |

Following the classic studies of Wiggers,[9] many workers have employed serial defibrillation in the open heart. However, Guyton and Satterfield found the average dog could usually be defibrillated through the unopened chest with a single shock of 160 volts, 60 cycle alternating current, and a shock of 220 volts was effective almost without fail. Our studies showed more consistent results with a single shock of higher voltage of 440 to 550 volts at 0.15 sec. This initial shock must be adequate to produce asystole, following which a faltering and then improving sinus rhythm develops (Fig. 1).

*Effect of Electric Current on the Heart.*—The dangers of producing burns with the use of high voltage on the exposed heart are apparent. Kaiser and coworkers,[10] using 130 to 230 volts at 0.1 and 0.25 sec., respectively, on the open heart, found microscopic burns in the myocardium of all dogs. However, only on those with the higher voltage and longer durations were the burns visible grossly and considered to be severe. Kirby and associates[11] reported similar findings. With a defibrillation shock of 220 volts, when the duration of the stimulus was increased from 0.1 to 0.5 sec., the hearts exhibited gross burns.

Although greater voltage is necessary when countershock is applied through the intact chest, it appears that the likelihood of damage to the heart is decreased.[12] Using a single countershock of 550 volts at 0.15 sec., no evidence of burns was seen on the skin, chest wall, or heart of any of our animals.

Three of the animals which survived 1 to 3 hours of fibrillation and subsequent defibrillation without sequelae were sacrificed from 2 to 3 months after the perfusion for examination of the heart. Gross examination revealed no evidence of scarring or healed burns of the epicardium or myocardium. However, histologic study* of random sections from the apex, septum, and base showed scattered focal and perivascular hemorrhages (Figs. 2*A* and 2*B*). In one of the hearts there were several areas of actual subendocardial infarction. In these lesions, there was gradual replacement of degenerating muscle fibers by proliferating granulation tissue. Occasional foreign body giant cells were

*Sections were kindly reviewed by Dr. K. P. Knudtson, pathologist, Veterans Administration Hospital, Seattle, Wash.

seen. In the absence of surface burns and due to their subendocardial location, it was thought these changes were most likely the result of hypoxia. The heart of a fourth animal which died 24 hours after a 3 hour and 20 minute period of perfusion showed gross areas of subepicardial hemorrhage as well as focal interstitial hemorrhage between muscle fibers consistent with acute hypoxia.

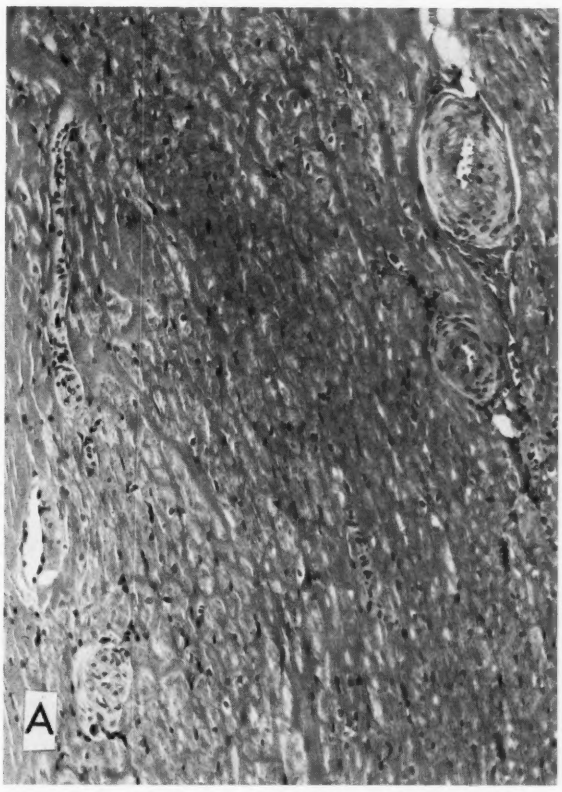


Fig. 2A.—(Photomicrograph) Dog No. 7 sacrificed at 2 months after perfusion. Heart grossly normal. Scattered focal and perivascular hemorrhage (×220; reduced ⅓).

*Perfusion.*—The vertical, all steel bubble oxygenator used for extracorporeal support in this study has been previously described.[13] The minimum perfusion rate necessary for prolonged support of the fibrillating heart and subsequent defibrillation and survival was unknown to us at the start of this work. From existing date, it was thought that flow rates varying from 45 to 70 c.c./Kg./min. should be adequate to maintain circulatory requirements. Based on the known values of cardiac output for dogs[14, 15] as the maximum desirable flow, and the report of Paneth, Lillehei, and co-workers[16] indicating the minimal flow requirement to prevent metabolic acidosis, we obtained moderate flow rates (Table II).

It was soon apparent that a compromise was necessary between desirable and obtainable flow rates. The principal factor limiting the rate of flow is the small size of the single peripheral venous catheter. The volume of venous return varied with the cross section of the catheter. The maximum obtainable venous return via the single peripheral femoral vein was found to average less

Fig. 2*B*.—Dog No. 8 sacrificed at 6 weeks. Heart grossly normal. Patchy degeneration of muscle fibers with gradual replacement by proliferating granulation tissue (×40; reduced ⅓).

TABLE II. PROLONGED VENTRICULAR FIBRILLATION

| EXPERIMENT | AVERAGE PERFUSION RATE (ML./KG.) | DURATION | SURVIVAL |
|---|---|---|---|
| 6 | 50 to 60 | 41 min. | Normal |
| 7 | 40 to 44 | 42 min. | Normal |
| 8 | 60 to 64 | 1 hr. 24 min. | Normal |
| 9 | 50 to 54 | 51 min. | Normal |
| 10 | 40 to 50 | 25 min. | Normal |
| 11 | 70 to 80 | 1 hr. 46 min. | Normal |
| 12 | 50 to 60 | 3 hr. | Normal |
| 13 | 65 to 70 | 2 1/2 hr. | Normal |
| 14 | 50 to 55 | 49 min. | Normal |
| 29 | 45 to 55 | 3 hr. 20 min. | Died |

than half the volume obtained by central cannulation and caval occlusion. Utilizing a double peripheral return from the jugular and femoral veins failed to add enough flow to warrant routine use. Gravity flow was unsatisfactory in maintaining whole body perfusion with peripheral cannulation and the heart

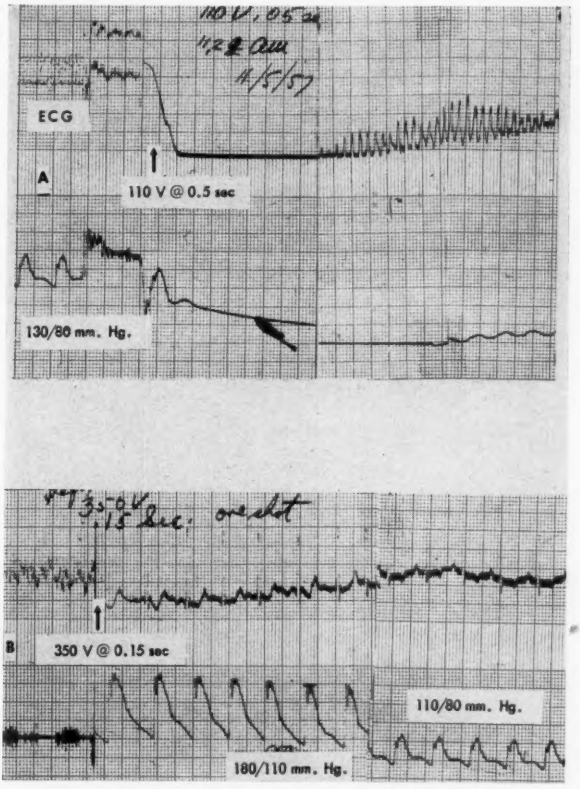


Fig. 3.—Experiment 8; 14 Kg. dog. *A*, Onset of ventricular fibrillation followed by 10 sec. of asystole (not shown). Fibrillatory pattern beginning with establishment of perfusion. *B*, Successful initial defibrillation after 1 hour and 24 minutes. Immediate resumption of sinus rhythm and peripheral pressure pattern. Change from 180/110 mm. Hg to 110/80 mm. Hg represents blood volume adjustment.

in fibrillation. Within several minutes of the beginning of perfusion the available venous return would seek its own flow level. Right heart pressure usually stabilized at 6 to 9 cm. of water. Best results in reaching a steady state were achieved when extracorporeal perfusion was begun within 20 to 30 seconds of the onset of ventricular fibrillation (Fig. 3). Further delay in establishing circulation profoundly affected the available venous return. Whether the electric shock produced a peripheral vascular dilatation, allowing pooling in postarteriolar vessels, or the venous run-off permitted pooling in certain venous

reservoirs, was not apparent. In the average run, a state of equilibrium was spontaneously established in which little or no adjustment of the pump speeds was necessary to balance the circulation during the 1- to 3-hour periods.

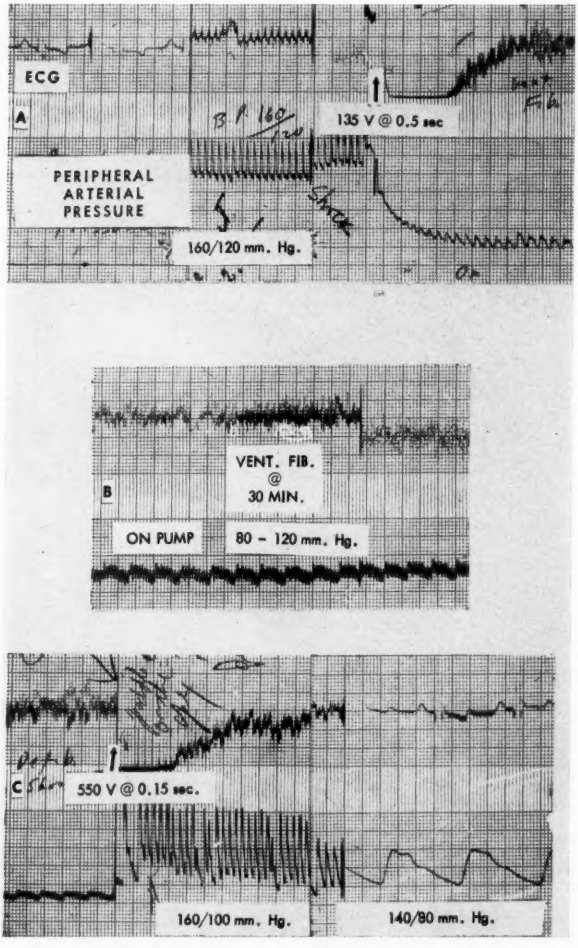


Fig. 4.—Experiment 7; 34 Kg. dog. *A*, Prefibrillation electrocardiogram and arterial pressure. Induction of ventricular fibrillation and prompt establishment of pump pattern on arterial tracing.

*B*, Constant fibrillatory pattern at 30 minutes. Mean arterial pressure from 80 to 120 mm. Hg at perfusion of 40 to 44 ml./Kg.

*C*, External defibrillation after 42 minutes. Postfibrillation electrocardiogram and blood pressure similar to prefibrillation values.

With flow rates, which were equivalent to one third to one half the animal's normal cardiac output, the mean arterial pressure varied from 80 to 100 mm. Hg (Fig. 4). Some animals exhibited a nearly normal blood pressure at these

low flow rates, whereas others maintained levels from 60 to 80 mm. Hg. It was found that the principal determinant of mean blood pressure was not flow rate but total peripheral resistance.[17]

*Biochemical Changes.*—The blood values incident to extracorporeal support with this particular oxygenator were reported previously. Detailed studies of blood chemistry or clotting factors were not a part of this work. For evaluation of the metabolic state after prolonged perfusion, venous pH was determined at intervals during several of the experiments. The results are given in Fig. 5. It may be seen that in most runs the venous pH remained within a fairly constant range. A cumulative acidosis was occasionally seen and usually responded to a slight increase in oxygen flow or perfusion rate, or both. In the last animal of the series, an effort was made to extend the perfusion period beyond 3 hours. At 3 hours and 20 minutes, although initial defibrillation was successful, the animal expired in 24 hours. Review of the pH revealed a marked acidosis with the lowest value in the series. The absence of cerebral activity prior to death suggested that the low flow rate resulted in cerebral hypoxia.

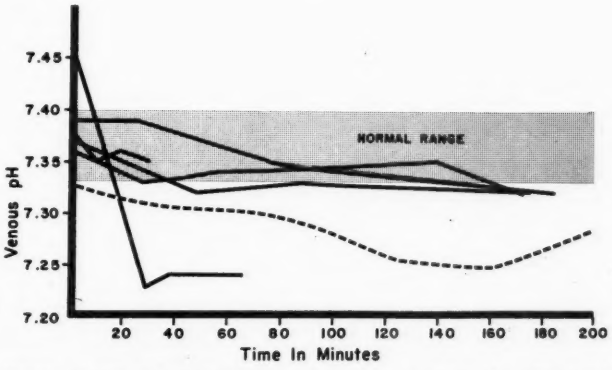


Fig. 5.—Graph of pH values during six fibrillation experiments with total body perfusion. Broken line indicates acute acidosis of the single nonsurviving animal.

It has been reported that the fibrillating heart loses potassium. In the isolated dog heart perfused with Locke's solution, Kehas and Hooker[18] noted the maximum value for potassium at ½ to 1 minute after the onset of fibrillation. Values at 3 to 4 minutes often showed a return to prefibrillation levels. In our studies, isolated determinations of serum potassium taken at intervals during several 1- and 2-hour fibrillation runs were unchanged from control values.

*Cardiac Activity During and After Ventricular Fibrillation.*—Prior to the development of extracorporeal pump-oxygenators, no direct measures were available for the support of coronary circulation during ventricular fibrillation other than cardiac massage. Observation of the electrocardiogram following the onset of ventricular fibrillation shows a fine, rapid, tremulous pattern of about 400 to 500 frequencies per minute. Without massage, direct coronary

perfusion or oxygenator support, the fibrillation pattern disintegrates, with progressive anoxia. After 3 to 5 minutes, the fine wave gives way to a slow, coarse wave of low amplitude, terminating in complete arrest. Experimental

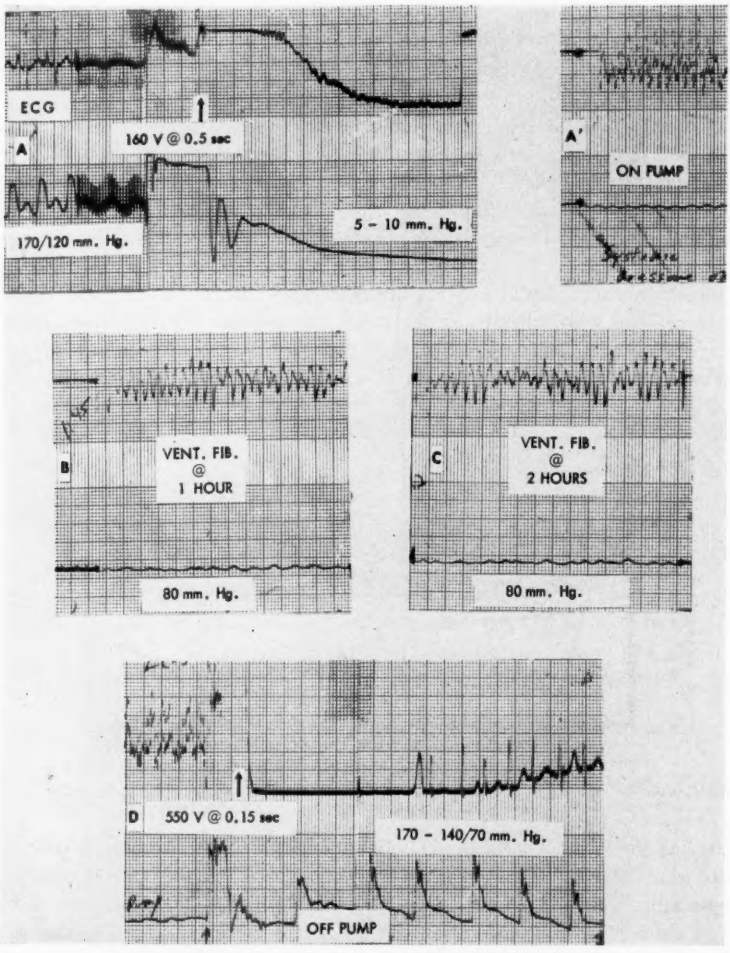


Fig. 6.—Experiment 12; 24 Kg. Dog. *A*, Closed chest induction of ventricular fibrillation. Ten to 12-second interval (not shown) prior to appearance of frank fibrillation and peripheral pressure (pump pattern) *A'*.

*B*, Essentially unchanging nature of ventricular fibrillation from *C*, hour to hour, suggesting satisfactory myocardial oxygenation. Mean arterial pressure 80 mm. Hg, perfusion 50 to 60 ml./Kg.

*D*, Electric defibrillation after 3 hours of extracardiac substitution. Countershock is followed by asystole and gradual resumption of sinus rhythm and prefibrillatory pressure tracing.

studies by Senning[19] with prolonged fibrillation in the normothermic, open chest animal showed that the fibrillatory pattern was unchanging during oxygenator support. Our findings were similar to those of Senning in that the wave pattern was consistent as long as mechanical difficulties in the system did

not intervene (Fig. 6). Countershock was not satisfactory for defibrillation unless threshold intensity was sufficient to produce asystole. Step increases in voltage were occasionally necessary in the larger animals. Following a brief asystole, a bradycardia with varying degrees of block was seen. During the subsequent 5 to 10 minutes, the rhythm gradually resumed the prefibrillatory pattern. In the surviving animals, systolic pressure was maintained at nearly normal levels without support. Ventilatory support was also unnecessary if cerebral centers had been adequately oxygenated. Spontaneous and effective ventilation began soon after discontinuance of the oxygenator. An effort was made to restore the animal to as nearly a normovolemic state as possible at the completion of the run. This was done by regaining the blood level in the oxygenator and observing the effect of hyper- or hypovolemia on the arterial pressure amplitude.

An interesting and unexpected finding was the presence of a regular atrial wave during the entire period of ventricular fibrillation. Although the atria would fibrillate when mechanically stimulated in the exposed heart, spontaneous rhythm was immediately resumed when the atria were released.

## DISCUSSION

In considering the deliberate use of ventricular fibrillation in human beings for open heart surgery, Glenn[20] has pointed out that coronary flow is uncontrolled and spontaneous defibrillation may occur with a well-oxygenated heart. Although we have not seen spontaneous defibrillation of the heart in normothermic dogs, Juvenelle,[21] Swan,[22] Bigelow[23] and others observed both spontaneous fibrillation and defibrillation during cooling and rewarming of the hypothermic dog.

Senning,[19] in 1951, was perhaps the first deliberately to induce ventricular fibrillation during total body perfusion. This suggestion was advanced in an effort to obtain cardiac standstill and to avoid coronary or cerebral air embolism during intracardiac surgery. In a subsequent monograph, Senning reported on many aspects of ventricular fibrillation. Using a modified Crafoord-Andersson-Björk disc oxygenator and central cannulation in the open chest animal, he was able to support the normothermic fibrillating heart for prolonged periods with successful defibrillation.

In a similar study but utilizing hypothermia with peripheral connection of the femoral vessels to the oxygenator, Juvenelle and co-workers[21] were able to support the fibrillating heart up to periods of 3 hours with subsequent defibrillation. Both Senning and Juvenelle were able to demonstrate that this arrhythmia is innocuous providing there is proper cardiac oxygenation.

In the absence of a massaging action by the contracting muscle, coronary flow may fluctuate to below or above normal values. Studies of oxygen consumption during fibrillation have been contradictory. Senning reported no significant difference in the fibrillating state as compared to the normally beating heart. On the other hand, Paul and co-workers[24] found slightly less oxygen utilization, while Green and Lorber[25] demonstrated an increased oxygen consumption.

The ease of defibrillation, both experimentally and clinically, while the heart is supported by the pump-oxygenator suggests the metabolism of the fibrillating and contracting heart is similar. As the oxygen requirement is certainly greater during fibrillation than for the heart in chemical or anoxic asystole, successful defibrillation and normal survival after prolonged extracorporeal substitution may indicate the effectiveness of a particular oxygenator and perfusion rate in meeting myocardial and cerebral demands.

## SUMMARY AND CONCLUSIONS

1. Prolonged substitution for the fibrillating ventricles has been accomplished in the closed chest animal by total body perfusion with a bubble oxygenator.

2. Defibrillation with electric countershock and restitution of sinus rhythm could be successfully achieved after long periods of fibrillation, providing the oxygen requirement of the myocardium is satisfied.

3. Coronary perfusion is necessary for myocardial support during the fibrillatory state.

4. Since deliberate ventricular fibrillation eliminates both the greater and lesser circulation, successful total body perfusion requires an efficient pump-oxygenator.

5. The controlled fibrillatory state is a useful experimental method for the study of hemodynamic phenomenon in the central circulation and the production of partial or total cardiogenic shock.

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# COMPARATIVE EFFECT OF 15 PER CENT POTASSIUM CHLORIDE AND 30 PER CENT POTASSIUM CITRATE IN RESUSCITATION FROM VENTRICULAR FIBRILLATION FOLLOWING ACUTE MYOCARDIAL INFARCTION

## An Experimental Study

*Fred Wasserman, M.D.,* Mark W. Wolcott, M.D.,** Curtis G. Wherry, M.D.,*** and Leonard Brodsky, M.D.,**** Coral Gables, Fla.*

THERE have been relatively few experimental observations on resuscitation of the heart following acute cyocardial infarction.[10, 11, 14, 15, 17] Previous work by one of us (F. W.) has demonstrated the relative effectiveness of 15 per cent potassium chloride in converting ventricular fibrillation to ventricular standstill in normothermic laboratory animals following acute myocardial infarction.[14] The restoration of coordinated cardiac beating occurred in all of these animals with variable difficulty. Other investigators have shown that 15 per cent potassium chloride would promptly convert ventricular fibrillation to ventricular standstill in deeply hypothermic laboratory animals without acute myocardial infarctions[13]; in these studies restoration of cardiac beating was apparently accomplished with greater facility using concentrated Ringer's lactate and molar sodium lactate.

Interesting case reports in the recent literature give dramatic testimony to what may be accomplished with active resuscitation attempts.[2, 5, 6, 8, 12] We believe as others[1, 18] that many deaths from acute myocardial infarction are "electrical" rather than "muscular," and it is this belief, supported in part by earlier observations in the experimental animal, that provided the impetus for the present study.

The purpose of this paper is to report on the relative efficacy of 15 per cent potassium chloride and 30 per cent potassium citrate in converting ventricular

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From The Veterans Administration Hospital and the University of Miami School of Medicine, Coral Gables, Fla.

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*Assistant Cardiologist, Assistant Director, Professional Services for Research, Veterans Administration Hospital, and Research Assistant Professor in Medicine, University of Miami School of Medicine, Coral Gables, Fla.

**Chief, Surgical Service, Veterans Administration Hospital, and Clinical Associate Professor of General Surgery, University of Miami School of Medicine, Coral Gables, Fla.

***Resident in Surgery, Veterans Administration Hospital, Coral Gables, Fla.

****Resident in Medicine, Veterans Administration Hospital, Coral Gables, Fla.

fibrillation to ventricular standstill following acute myocardial infarction in the experimental animal. The ultimate aim of this effort is to standardize a procedure which can be instituted with sufficient ease and frequency to justify organized and vigorous efforts in attempts to resuscitate patients dying from coronary artery disease.

## METHOD AND MATERIAL

Forty mongrel dogs weighing between 15 and 40 pounds were studied. The typical animal was lightly anesthetized with pentobarbital sodium and given positive pressure air and/or oxygen through an endotracheal tube; both mechanical respirators and bagging procedures were employed. Each experiment was performed utilizing strict aseptic technique. The chest was opened with a transsternal incision to enhance exposure. Hemostasis was carefully maintained. The femoral artery was cannulated for the withdrawal of blood specimens (pH, serum electrolyte, and chemical determinations) and the recording of arterial blood pressures. Continuous electrocardiograms and simultaneous arterial pressures were recorded on the Sanborn Twin Visocardiette. The femoral vein was cannulated for the administration of various drugs. The anterior descending branch of the left coronary artery was ligated with a 1-0 silk suture, 0.5 cm. distal to its bifurcation. Electrocardiographic tracings were followed until the changes characteristic of an acute myocardial infarction were observed. Subsequently, ventricular fibrillation either (a) occurred spontaneously within 10 to 15 minutes following the acute infarction or (b) was induced by electric shock (50 to 150 volts) applied directly to the heart, utilizing a defibrillator. Untreated ventricular fibrillation occurring under these circumstances was irreversible.

Immediately after the onset of ventricular fibrillation 20 animals were treated with 15 per cent potassium chloride to induce ventricular standstill and 20 animals were given 30 per cent potassium citrate. The effect of the intracardiac administration of hypertonic potassium (into one or both ventricles) was compared to the administration of the solutions into the proximal portion of the cross-clamped aorta and into one or both ventricles with the aorta cross-clamped.

Following the administration of potassium, slow cardiac massage (30 to 50 per minute) was initiated until the electrocardiogram demonstrated conversion of the ventricular fibrillation to ventricular standstill. The effectiveness of blood flow in the large arteries could roughly be gauged by the pressure recordings from the femoral artery. With the onset of ventricular standstill, cardiac message was speeded to 70 to 100 times per minute, and one molar sodium lactate was administered intravenously and occasionally in small amounts (up to 20 c.c.) into the heart. The total amount administered ranged from 30 to 350 c.c. (see Table I). In each animal studied, repeated episodes of ventricular fibrillation were terminated with hypertonic potassium chloride or potassium citrate, depending upon which agent was being studied

with that particular animal. Restoration of cardiac beating following each episode of ventricular fibrillation in the same animal was approached, utilizing a method similar to that employed for the initial episode. Where cardiac message or spontaneous cardiac beating appeared to be unable to maintain a satisfactory level of arterial pressure, varying concentrations of intravenous l-norepinephrine were administered. Following successful restoration of cardiac beating and return of the blood pressure to near normal levels, the chest was closed with wire stay sutures and silk, and the animals were gradually weaned from the artificial respirator. The femoral catheters were subsequently removed, 10 to 15 minutes following the appearance of normal sinus rhythm associated with a normal or near normal arterial pressure. Postoperative animals were given intramuscular crystalline penicillin and streptomycin. No intravenous fluids were administered in the postoperative period; the animals were, however, allowed to eat and drink ad libitum. Frequent samples of blood for pH and serum electrolytes were analyzed using standard laboratory procedures to compare the electrolyte, chemical, and pH changes with 15 per cent potassium chloride and 30 per cent potassium citrate induced arrest.

## RESULTS

Ventricular fibrillation, complicating acute myocardial infarction, in the experimental animal represents an irreversible terminal mechanism.

*Changes in Arterial pH, Serum Electrolytes in Chemistry.—*

1. *15 per cent potassium chloride:* Two 5 to 10 c.c. intracardiac injections of 15 per cent potassium chloride were given to terminate each episode of ventricular fibrillation. The total amount of potassium chloride administered was equally divided between the right and left ventricular cavity when intracardiac injection was studied. When the aorta was cross-clamped the total amount was given into the proximal portion of the aorta. It appears that conversion from ventricular fibrillation to ventricular standstill was accomplished with equal facility with both methods of administration. The total amount of potassium chloride required to induce ventricular standstill, following each episode of ventricular fibrillation, averaged 15 c.c. (see Table I).

Electrolyte and pH changes: The serum potassium concentrations attained their maximum immediately following the administration of potassium chloride. These very high levels (required to produce ventricular standstill) ranged from 8.4 mEq. per liter to 40.5 mEq. per liter. With the restoration of cardiac beating, high serum potassium levels fell precipitously, usually within 5 to 15 minutes, to levels ranging from 4.8 mEq. per liter to 10.8 mEq. per liter. As the effectiveness of cardiac beating improved, elevated serum potassium concentrations gradually returned to levels near normal or only slightly higher than normal (6.9 mEq. per liter).

Subsequent episodes of ventricular fibrillation, terminated with potassium chloride, were followed by an abrupt elevation in the serum potassium, usually to a concentration slightly more than that observed following termination of the initial episode. The serum chloride level showed a marked increase as a result of the hypertonic potassium chloride arrest (paralleling the serum potassium). The serum sodium concentration gradually increased following the intravenous administration of molar sodium lactate and reached its zenith toward the end of the experiment. Maximum serum sodium levels ranged from 138 mEq. per liter to 216 mEq. per liter. The serum calcium level decreased slightly throughout the experiment; the low range being 8.2 mg. per cent to 8.7 mg. per cent. The carbon dioxide combining power decreased abruptly following induced cardiac arrest (Fig. 1*B*) and subsequently returned to near normal levels. The average fall was 14 mEq. per liter. There was no significant change in the serum magnesium concentration. The arterial pH fell significantly. The most abrupt fall occurred immediately after the administration of potassium chloride and in general, coincided with the onset of ventricular standstill. Subsequently, the blood pH either remained low, in spite of the administration of molar sodium lactate, or was decreased further by an unknown, apparently paradoxical, mechanism. The average fall of the arterial pH was 0.32. Fig. 1*A* and 1*B* show the significant mean changes in the serum sodium, potassium, carbon dioxide combining power and arterial pH occurring during the procedure.

2. *30 per cent potassium citrate:* In 20 animals studied with 30 per cent potassium citrate arrest, the most effective method of administration appeared to be equal division of the total amount into both the right and left ventricle. The total amount of potassium citrate required to induce ventricular arrest (standstill) ranged from 4 c.c. to 32 c.c.

Electrolyte and pH changes: In general, the changes in the serum electrolytes corresponded to those observed following hypertonic potassium chloride with several exceptions. The serum chloride level remained essentially unchanged following potassium citrate arrest, and the increase in the serum potassium level was never as marked as that observed following potassium chloride arrest. The maximum serum potassium concentrations ranged between 10.1 and 19.5 mEq. per liter. The carbon dioxide combining power decreased slightly throughout the procedure (as with potassium chloride), and the serum calcium either remained unchanged or decreased slightly, following, and probably as a result of, the intravenous sodium lactate therapy.[4] There was no apparent change in the serum magnesium. Comparative mean electrolyte and pH changes are shown in Figs. 1*A* and 1*B*.

***Effect of 15 Per Cent Potassium Chloride and 30 Per Cent Potassium Citrate on Resuscitation From Ventricular Fibrillation.***—

The mean number of episodes of ventricular fibrillation using hypertonic potassium chloride arrest was 5; with hypertonic potassium citrate arrest, this was reduced to 3.

In 39 of the 40 animals studied, the intracardiac administration of both hypertonic potassium solutions converted the ventricular fibrillation to ventricular standstill; one animal died an anesthetic death. In general, hypertonic potassium citrate appeared to be more effective in that ventricular

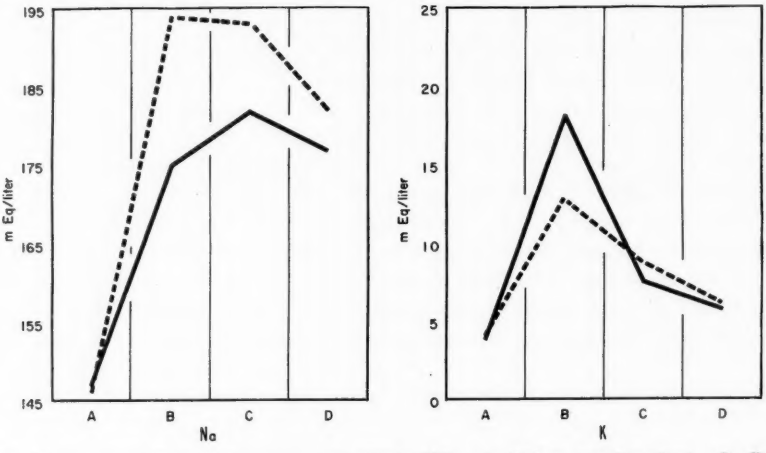


Fig. 1*A*.— ——— K Cl 15%. - - - - - K citrate 30%. *A*, Before opening chest. *B*, Cardiac arrest. *C*, Restoration of cardiac action. *D*, End of procedure.

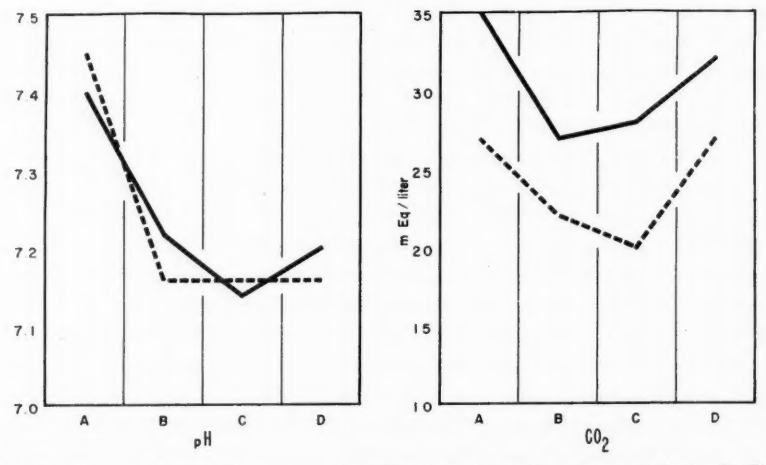


Fig. 1*B*.— ——— K Cl 15%. - - - - - K citrate 30%. *A*, Before opening chest. *B*, Cardiac arrest. *C*, Restoration of cardiac action. *D*, End of procedure.

standstill occurred more promptly and with greater facility; the tendency to develop multiple episodes of ventricular fibrillation was reduced with 30 per cent potassium citrate (Table I).

In 19 of the 40 animals, we were able to restore the heart to normal sinus rhythm and the blood pressure to normal or near normal ranges (Fig. 2).

TABLE I

| TYPE OF K SOLUTION USED | AVERAGE AMOUNT K TO TERMINATE EACH EPISODE OF VENTRICULAR FIBRILLATION | AVERAGE NO. OF EPISODES OF VENTRICULAR FILBRILLATION | AVERAGE DURATION OF INITIAL EPISODE OF VENTRICULAR FIBRILLATION | AVERAGE AMOUNT MSL* AFTER EACH EPISODE OF ARREST |
|---|---|---|---|---|
| 15% KCl | 15 c.c. | 5 | 3 min. | 100 c.c. |
| 30% K Citrate | 5 c.c. | 3 | 50 sec. | 40 c.c. |

*Molar sodium lactate.

Twelve of these subsequently died while the chest was being closed. Six of the 12 animals died as a result of the following difficulties: hemorrhage (1 dog); apparently irreversible damage to the respiratory center (manifested by an inability to resume breathing spontaneously [2 dogs]); trauma and laceration of the myocardium incident to massage (2 dogs); and inadequate pulmonary ventilation following closure of the chest (1 dog). Seven animals survived the entire procedure, living from 1 hour to 6 months. The hearts of these animals were subsequently examined grossly and microscopically.

In the remaining 20 animals, normal sinus rhythm never became re-established. In many of these, an auriculoventricular nodal rhythm or a transient return to normal sinus rhythm was observed, following which repeated episodes of ventricular fibrillation signified the end of the experiment.

*Pathologic Examination of the Heart.—*

Pathologic examination of the hearts at varying periods of time following successful cardiac resuscitation showed either the change of an acute or chronic myocardial infarction. In an animal sacrificed after surviving 6 months, the large anterolateral myocardial infarction visualized during the acute procedure had healed so that only the residual changes of a small transmural area of fibrosis in the anterior wall of the left ventricular myocardium was demonstrated histologically.

## DISCUSSION

Previous experimental work had demonstrated that in spite of an acute, apparently extensive, myocardial infarction, 3 out of 4 animals studied with terminal ventricular fibrillation were ultimately restored to a supraventricular rhythm with normal or near normal blood pressures.[14] In the present study, performed utilizing strict aseptic technique, earlier observations (using hypertonic potassium chloride) were repeated and compared with hypertonic potassium citrate arrest. From this study it appears that 30 per cent potassium citrate is superior to 15 per cent potassium chloride in converting ventricular fibrillation to cardiac standstill. Induced potassium citrate arrest resulted in less severe potassium intoxication which appeared to be more readily reversed by treatment with molar sodium lactate.[3] Apparently undesirable hyperchloremia observed with potassium chloride was not present when potassium

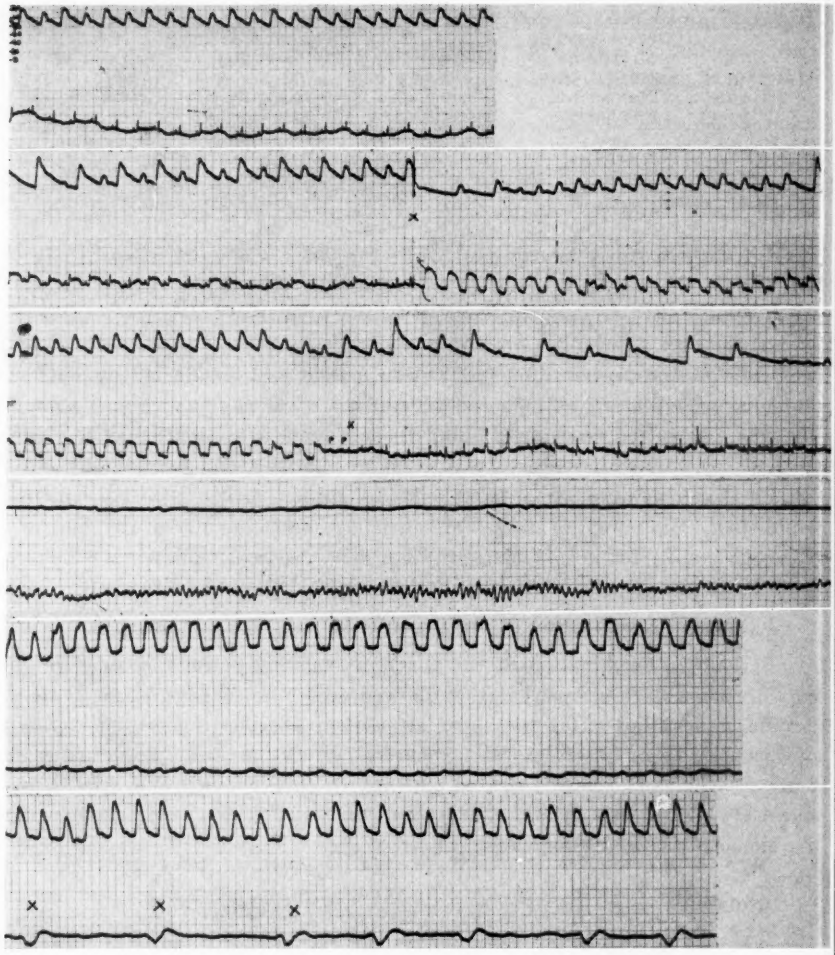


Fig. 2.—*A*, Control (Lead II); simultaneous arterial pressure is 120/80 mm. Hg.

*B*, First half of record taken 10 minutes after ligation of the anterior descending coronary artery shows RS-T segment elevation of the type seen in acute coronary artery occlusion. The arterial pressure is 124/60 mm. Hg. From *X* until the end of the record, note the characteristic high take-off of the RS-T segment accompanying the acute myocardial infarction. The pressure has fallen to 84/44 mm. Hg.

*C*, Thirty minutes after acute coronary ligation note the conduction disturbance at *X*.

*D*, Forty minutes following acute coronary artery occlusion at 2.30 P.M., ventricular fibrillation was induced by applying electrodes to the ventricles and administering a single 50-volt electric shock.

*E*, Forty-five seconds after 3 c.c. of 30 per cent potassium citrate had been given into both ventricles; ventricular fibrillation has been converted to ventricular standstill. Cardiac massage was started (at a rate of 100 per minute). Note the apparent effectiveness of arterial flow as recorded by a catheter in the femoral artery.

*F*, Three minutes after ventricular standstill, note the onset of electrical activity at *X* with widened, bizarre QRS complexes. Intravenous molar sodium lactate has been started at a rate of 5 c.c. per minute.

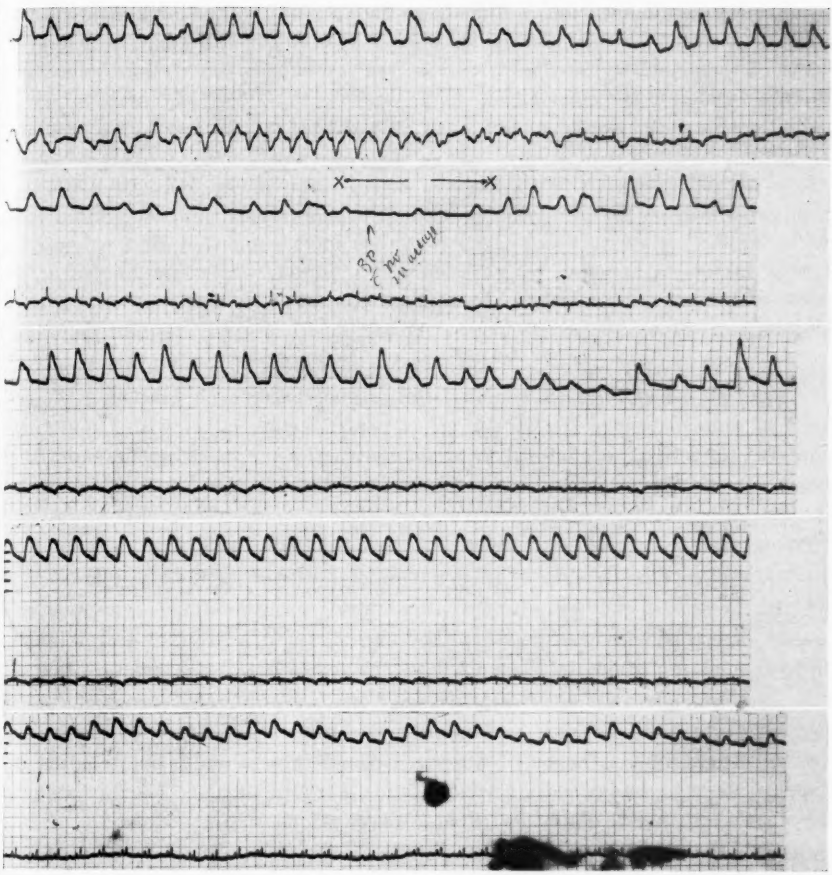


Fig. 2.—Cont'd. *G*, Five minutes after *F*, following a total of 40 c.c. of molar sodium lactate, there is progressive narrowing of the QRS complexes and a suggestion of restoration of auricular activity (*P*). Cardiac massage has been continuous.

*H*. Twelve minutes after ventricular standstill, the QRS complexes have narrowed to a near normal width. Note the effects (*X*) on the arterial pressure of discontinuing cardiac massage.

*I*, Fifteen minutes after induced ventricular arrest, there is a return to normal sinus rhythm. The arterial pressure is being supported with a very slow intravenous drip containing 1 ampule of l-norepinephrine diluted in 40 c.c. molar sodium lactate.

*J*, Twenty-five minutes after ventricular standstill, there is spontaneous effective cardiac beating (without massage). The arterial pressure is maintained at 110/60 mm. Hg without massage or drugs. The QRS complexes are low in amplitude. The T-wave is inverted and the QT interval appears prolonged for the heart rate (approximately 116 per minute).

*K*, One hour and thirty minutes after "death" in ventricular fibrillation, the arterial pressure is 100/60 mm. Hg. The chest has been closed and the animal has resumed spontaneous breathing.

citrate was used. The paradoxical observation that the arterial pH continued to remain low with both hypertonic potassium solutions, even after restoration of effective cardiac beating, is difficult to understand; particularly after rather large amounts of a mild alkalizing solution (sodium lactate) had been given. In the hope of clarifying this, we are at the present time studying the carbon dioxide tension in the plasma.

Changes in the serum calcium probably reflected the use of varying amounts of sodium lactate intravenously as has been previously noted.[4] This decrease in the serum calcium would appear to increase the cardiotoxic effect of even a slight elevation in the serum potassium concentration, and we should like to be able to prevent this. Earlier studies have suggested that calcium is useful in hearts arrested by potassium.[9, 16] In our experience, however, it seemed difficult to quantitate the amount of calcium which was helpful and the danger of overdosage (recurrent ventricular fibrillation) limited its usage.

*Blood Pressure Changes.*—The persistent acidosis might explain the tendency for some of the animals to remain mildly hypotensive even following the return of a normal sinus mechanism (electrocardiographically) with apparently forceful, effective, mechanical cardiac contractions. In these animals, the combination of l-norepinephrine and molar sodium lactate often successfully restored the blood pressure to normal or near normal levels. One might anticipate such a reaction as a result of the observation that alkalosis appears to enhance the effectiveness of intravenous vasopressor agents.[7]

In the animal surviving longest (6 months), an interesting, perhaps superfluous, observation is the fact that this dog delivered a litter of 4 apparently normal pups approximately 5 months after successful cardiac resuscitation.

From our observations it appears that one can anticipate occasional long-term survival animals with a standardized resuscitation attempt utilizing 30 per cent potassium citrate to induce ventricular arrest and a combination of cardiac massage, molar sodium lactate, and l-norepinephrine. Further studies have been initiated to elaborate on these observations.

## SUMMARY

Thirty per cent potassium citrate was more effective than hypertonic potassium chloride in converting otherwise irreversible ventricular fibrillation to ventricular standstill in normothermic laboratory animals with acute myocardial infarction. Continuous electrocardiograms and arterial pressures were recorded throughout each procedure and serial electrolyte, chemical, and pH changes were studied and are discussed.

Of the 40 animals included in this report, 7 survived and were observed from hours to months following successful cardiac resuscitation.

Some of the problems encountered in this study are presented. The need for further experimental observations in organized resuscitative attempts following death from acute myocardial infarction is stressed.

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# TIME STUDIES OF ACUTE, REVERSIBLE, CORONARY OCCLUSIONS IN DOGS

*Seiichi Yabuki, M.D.,* Gumersindo Blanco, M.D.,** Joseph E. Imbriglia, M.D.,*** Lamberto Bentivoglio, M.D.,**** and Charles P. Bailey, M.D.,***** Philadelphia, Pa.*

THE surgical management of coronary occlusion has been directed toward relief of chronic myocardial ischemia by means of a number of revascularization procedures. In the past, the indirect approach to revascularization has predominated with grafting of various tissues onto the myocardium[1, 2] (omentum, lung, pectoral muscle), implantation of systemic arteries into the heart musculature,[3] abrasion of the epicardium,[4] and arterialization,[5] or merely constriction of the coronary sinus, to name a few. Lately, the direct attack on the constricting lesion, whether by endarterectomy,[6] or by excision and replacement of the diseased segment,[7] has made its appearance both clinically and experimentally.

Once the feasibility of the direct approach to the well-established occlusion is accepted, it takes but an additional step to consider the applicability of similar direct measures on the acutely obstructing process, that is, the emergency surgical treatment of the acute coronary occlusion.

An essential factor in establishing the effectiveness of such a policy would be to ascertain the available time during which emergency relief of an acute complete occlusion would result in preventing death of the myocardium. Should this time be commensurate with the interval needed for the rendering of a correct clinical diagnosis, transportation of the patient to a surgical facility and the performance of an emergency operation, such a policy would have a rational basis.

In order to determine the critical period during which emergency relief of the complete occlusive process would be curative, the following experiments were undertaken.

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*Fellow, Department of Thoracic Surgery, Hahnemann Medical College and Hospital, Philadelphia, Pa.

**Director, Cardiac Surgery Research, Mary Bailey Foundation for Heart and Great Vessel Research, Philadelphia, Pa.

***Chairman, Department of Pathology, Hahnemann Medical College and Hospital, Philadelphia, Pa.

****Fellow, Department of Thoracic Surgery, Hahnemann Medical College and Hospital, Philadelphia, Pa.

*****Director, Bailey Thoracic Clinic, Philadelphia, Pa.

METHOD

Mongrel dogs weighing between 12 and 20 Kg. were anesthetized with intravenous Pentothal, intubated, and attached to a mechanical respirator. One hundred per cent oxygen was administered throughout the procedure. The left common carotid artery was cannulated and continuous blood pressure was recorded thereby.

A left thoracotomy was performed through the fifth intercostal space. After incising the pericardium and securing it to the chest incision, the anterior descending branch of the left coronary artery was dissected free in

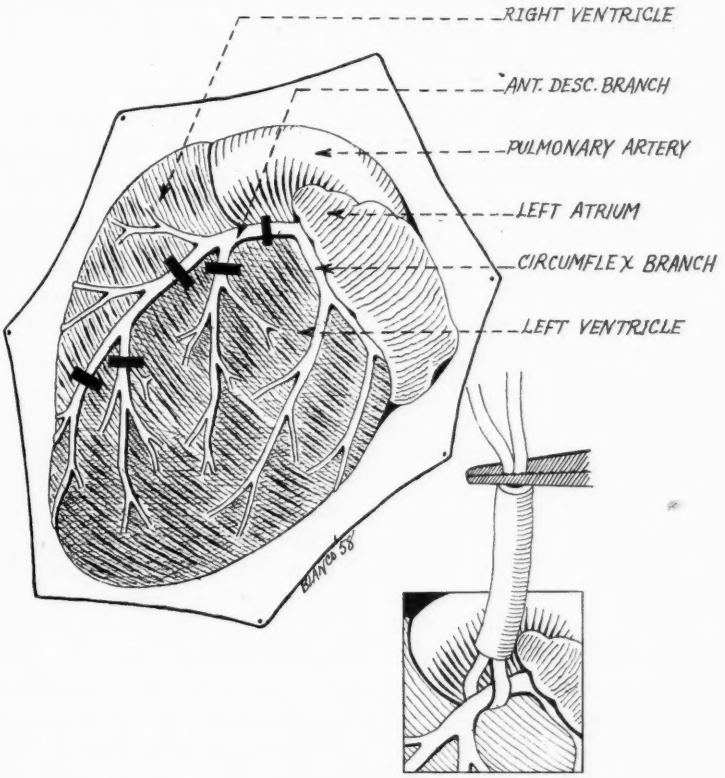


Fig. 1.

its proximal 0.5 to 1.5 cm., care being taken to avoid trauma to its branches or to the accompanying veins. A No. 2 braided silk ligature was then passed around the artery and both ends were threaded through a short length of soft plastic tubing in order to provide a tourniquet.

In 7 of the dogs the same procedure was employed at other sites along the course of the anterior descending artery or its two main branches, as indicated in Fig. 1.

The tourniquet was then tightened, occluding the involved portion of the artery completely, as evidenced by distal collapse of the vessel and appearance of a cyanotic area corresponding to its distribution. Stimulation of the heart was avoided during the occlusion period, but undue drying of the myocardial surface was prevented by moistening it with warm saline solution.

A record was kept of the myocardial action and color, blood pressure, and condition of the corneal reflex. After a definite period, the occlusion was terminated, the silk ligature was removed, and the pericardial and chest incisions were closed. If fibrillation occurred during the period of occlusion the tourniquet was released and removed. Massage and electrical defibrillation were instituted and, after the return of a normal heart rate and rhythm and stabilization of blood pressure, the operation was terminated in the manner previously described.

Electrocardiogram recordings of the three standard leads were obtained before opening the chest, during and after occlusion, and after chest closure. They were repeated at the time of sacrifice which varied between 48 hours and 14 days after the operation.

At sacrifice, the heart was excised and examined grossly. Twenty hearts were examined microscopically, noting the condition of the myocardium supplied by the temporarily occluded artery, and examining the vessel itself for evidence of permanent damage or of postoperative thrombotic phenomena. The latter were reported only once.

## RESULTS

As described under Method, the area of heart muscle supplied by the occluded vessel became cyanotic within 2 to 3 minutes after starting the occlusion. As time went on, the cyanosis deepened and the affected area became sharply delineated from the surrounding normal myocardium. In some of the longer occlusions (over 45 minutes), paradoxical movement of the cyanotic area was observed with bulging during systole. After release of the occlusion, the distal portions of the artery were seen to fill readily from above and the cyanotic discoloration disappeared gradually.

*Fibrillation.*—Fourteen (46.7 per cent) out of 30 dogs exhibited ventricular fibrillation. In 9 (30 per cent), fibrillation appeared during the occlusion at which time the latter was terminated. The time of onset of fibrillation averaged 9 minutes and 36 seconds (range—from 1 to 20 minutes). Five (16.7 per cent) hearts fibrillated upon release of the constricting ligature after a predetermined period of occlusion. One dog died. The rest were successfully defibrillated with application of cardiac massage and one or more electric shocks.

Electrocardiographic findings during occlusion were predominantly T-wave and RS-T segment changes. During occlusion, inversion of the T wave occurred frequently, but in 3 dogs, with inverted T waves before occlusion, we observed positive deflections shortly after occlusion. Marked shifts of RS-T

segments were noted on 11 dogs, with depression in 5 and elevation in 6. In the majority, these changes disappeared and the deflection returned to the preoperative state upon the release of the constricting ligature.

Electrocardiographic changes at sacrifice (2 to 14 days after operation) in all dogs, with temporary occlusion periods over 45 minutes, showed evidence of severe coronary insufficiency or of definite anterolateral infarction. In animals with shorter occlusive periods, the findings were minimal or absent, with the exception of one dog with a 12-minute occlusion in which changes suggestive of myocardial infarction were reported. This animal had suffered from ventricular fibrillation and received multiple electric shocks to restore normal rhythm. At autopsy, there was hemorrhage around the area of ligation but no microscopic evidence of infarct. The pathologic changes were probably caused by the trauma incidental to the resuscitative measures.

*Pathologic Changes Following Temporary Ligation.*—Twenty hearts were examined, 2 to 14 days after operation. Five dogs undergoing occlusion of branches of the anterior descending coronary artery for 30 minutes did not show gross or microscopic evidence of myocardial involvement.

In 15 dogs, there were definite myocardial changes as follows: All hearts subjected to occlusion of the anterior descending branch of the left coronary artery for 45 minutes or more showed myocardial necrosis with varying degrees of round cell infiltration or fibrous replacement. With one exception, the coronary vessels were patent at the time of sacrifice. In this animal (No. 27) the anterior descending artery showed partial occlusion with thrombus at the site of temporary ligation. This obstruction had narrowed the lumen of the vessel to 25 per cent of normal. Microscopic findings in dogs undergoing occlusions of 30 minutes' duration or less were minimal and were limited to hemorrhages and inflammatory cell infiltration. Invariably these changes occurred in dogs with ventricular fibrillation at some time during the procedure, and were undoubtedly the result of trauma during cardiac massage and electric shock.

Pericarditis was reported in 7 dogs but appeared unrelated to myocardial damage.

## DISCUSSION

Our operative, electrocardiographic and pathologic findings parallel the reported experiences of others with this type of vascular insult to the myocardium.

The incidence of ventricular fibrillation in this group (46.7 per cent) is lower than has been reported by Bakst,[8] Orias,[9] or Litvak[10] (60.6 per cent to 78.6 per cent) but higher than the 10 per cent encountered by Chardack[11] who also used 100 per cent oxygen given by an artificial respirator during the procedure.

The phenomenon of fibrillation at the time of re-establishment of circulation has been observed by others (Blumgart[12] and Sewell[13] and their coworkers) and has been explained by Beck[14] on the basis of a sudden change

in oxygen potential of adjacent areas of myocardium. There was only one death from this arrhythmia attesting to the efficacy of prompt cardiac massage and electric defibrillatory shock.

The electrocardiographic findings for this type of occlusion have been extensively studied and the typical T wave and RS-T segment changes have been emphasized by Bayley and associates,[15] Harris and Hussey,[16] Rakita,[17] and Kimura.[18] It would be interesting to determine the changes incidental to partial occlusion in objectively graded degrees, and by the same method to determine the sequelae of partial occlusions from a pathologic standpoint inasmuch as, in clinically encountered occlusion, partial obstructions of one or more segments of an artery are probably very frequent.

The pathologic evidence shows that a complete occlusion of a major branch of a coronary artery (the anterior descending in these experiments) is associated with death of cardiac muscle if the occlusion lasts for 45 minutes or more. This is in accord with the studies of Blumgart[19] and co-workers and would vitiate the advantages of direct attack on the acutely obstructed artery from the standpoint of preventing muscle death, unless such an attack could be carried out during the "critical" period. Relief of a complete obstruction might still be beneficial in reducing the irritability of still viable but anoxic myocardium about the central area of necrosis.

Again, the problem of the partial obstruction and the emergency surgical efforts to relieve it and/or to prevent it from becoming complete is quite different and out of the scope of this paper.

## CONCLUSION

A time study was undertaken to determine the period during which direct relief of a complete obstruction to a main branch of a coronary artery would prevent death of the portion of heart muscle supplied by the involved artery. The evidence presented indicates that a complete occlusion lasting for more than 45 minutes will produce the irreversible changes characteristic of myocardial infarction.

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# OXYGEN CONSUMPTION DURING CARDIOPULMONARY BYPASS

*Albert Starr, M.D., Portland, Ore.*

MUCH attention has been directed to studies of oxygen consumption during cardiopulmonary bypass, both in dogs and human beings. The difficulty of such studies is attested to by the variety of different results obtained by other investigators.

Attention is directed in the present study to the factors relating to oxygen consumption during extracorporeal circulation at varying perfusion rates in dogs and at flows of basal cardiac output in human beings.

## METHODS

*Part I. Dogs.—*

Fifty-seven mongrel dogs were placed on total cardiopulmonary bypass with a gravity drainage rotating disc oxygenator for a minimum of one hour, under light Pentothal anesthesia, at varying flows. Oxygen consumption was measured in 25 animals by means of a Douglas bag just prior to thoracotomy. Operation was carried out via a right thoracotomy through the fourth interspace with cannulation of the superior vena cava, by way of the azygous vein, and inferior vena cava, by way of the right atrial appendage. The aorta was cannulated via the right carotid artery. The partial pressure of oxygen in the arterial blood was monitored by means of a Clark electrode polarographic circuit and was not allowed to rise above 200 mm. Hg. Arterial oxygen saturation was always over 90 per cent. Various intracardiac procedures were carried out, such as creation and closure of interatrial and interventricular septal defects which were not considered to interfere with metabolic studies.

Arterial and venous samples, drawn simultaneously during perfusion, were analyzed by the Van Slyke technique for oxygen content, oxygen capacity, and carbon dioxide content. Flows were measured following perfusion using the pump settings noted during perfusion and the same arterial catheter as used during perfusion. The validity of this method has been demonstrated by others and ourselves, when a completely occlusive pump is used.[1] The flow was kept constant for a minimum of 15 minutes prior to sample to ensure a steady state. One hundred and twenty observations were made on this group of 57 dogs. Oxygen consumption was calculated from the arterial-venous oxygen difference and flow. Rectal and blood temperatures were recorded for each oxygen determination and were always between 36° and 37° C. This was accomplished

From the Crippled Children's Division and Department of Surgery, University of Oregon Medical School, Portland, Ore.

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by means of a low spectrum infrared heater described in another publication. The electrocardiogram and arterial and inferior vena cava pressures were monitored on a multichannel Sanborn recorder.

Dog surface area was calculated by the following formula: surface = 1.12 weight$^{2/3}$ (see Ref. 2). Variations in arterial pressure, venous oxygen saturation, and oxygen consumption were then related to flow in liters per square meter surface area per minute.

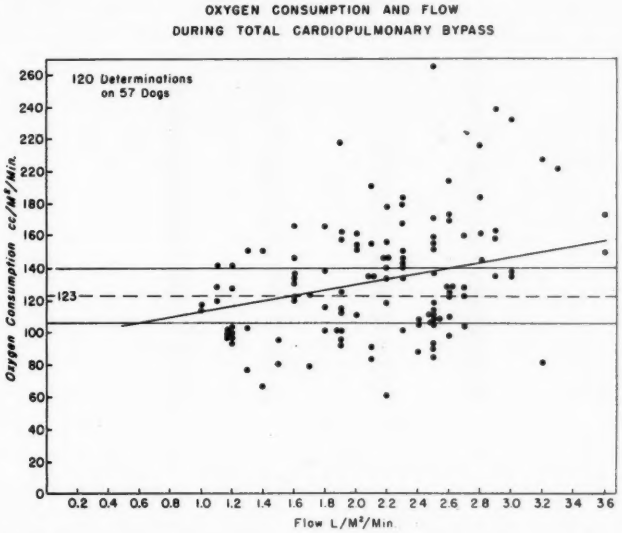


Fig. 1.—Relationship between oxygen consumption and flow described as a straight line function.

$\hat{y} = 96.50 + 16.89\ x.$

$sy \cdot x = 28.74,\ r^2 = 0.105.$

t for slope = 2.54, $P < 0.05$.

The horizontal dotted line represents the mean oxygen consumption of 25 anesthetized dogs with the line above and below indicating two standard deviations.

*Part II. Human Beings.*—

Similar data were obtained from the first 15 consecutive patients operated upon at the University of Oregon Medical School Teaching Hospital. A flow approximating basal cardiac output was maintained constant throughout the perfusion period, utilizing the same apparatus as in the dog studies.

An arterial sample was drawn from the patient 10 minutes prior to the onset of cardiac bypass. At the 5-minute mark, following the onset of perfusion, and at 20-minute intervals thereafter, arterial and venous samples were drawn from the arterial end plate and venous reservoir, respectively. An arterial sample was drawn 10 minutes following the cessation of cardiac bypass. In addition to oxygen and carbon dioxide determinations using the Van Slyke method, pH was measured with a Cambridge research model pH meter with a constant temperature air bath at 37° C. and arterial buffer base calculated from the Singer-Hastings normograms.[3]

RESULTS

*Part I. Dogs.—*

As flow is increased in the entire range studied, oxygen consumption increases as demonstrated in Fig. 1. This rise in oxygen consumption continues as flow is increased above levels now in clinical use in many hospitals.

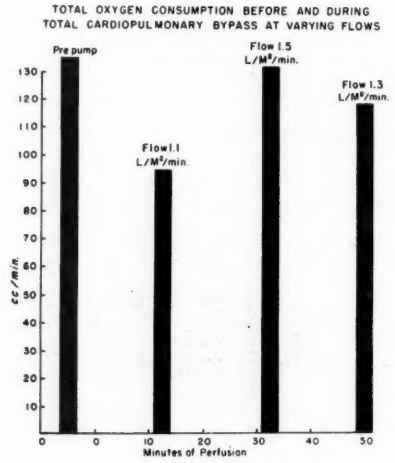


Fig. 2.

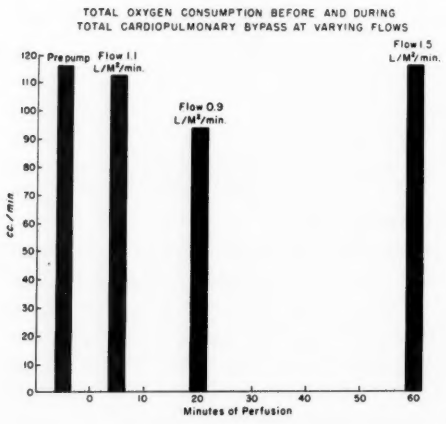


Fig. 3.

Figs. 2 and 3.—These figures demonstrate that changes in oxygen consumption will follow the same direction as changes in flow in a single animal.

The mean oxygen consumption prior to cardiac bypass but following tracheal intubation of 25 dogs in this laboratory is 123 c.c. per minute per square meter of body surface. When oxygen consumption during bypass is

compared to this value, it is noted that at flows below 1.6 L. per square meter of body surface there is greater tendency for oxygen consumption to be lower than the pre-perfusion level, and similarly, at flows above this value, there is greater tendency for the oxygen consumption to be higher than the pre-perfusion value.

As noted from Fig. 1, there is great variation in oxygen consumption in different dogs at the same flow rate. This is probably related to variations from the basal state, perhaps due to variations in the level of anesthesia. All animals were similarly anesthetized with Pentothal sodium but it was noted

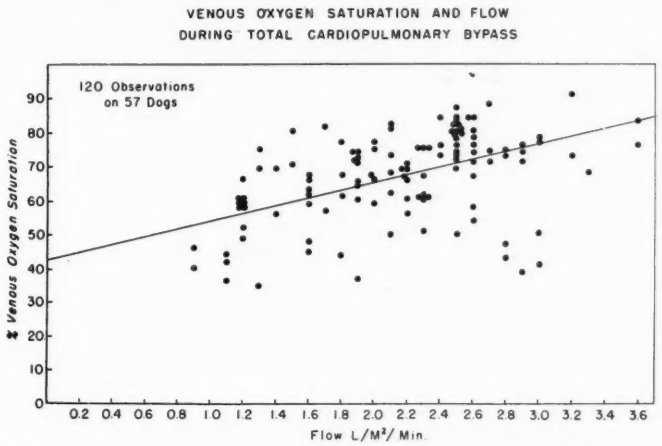


Fig. 4.—This figure demonstrates that venous oxygen saturation is well described by a linear function of flow and increases with increasing flow. It is noteworthy that venous oxygen saturation of 70 per cent is obtained at a flow of 2.4 L./M.²/min.

$\hat{y} = 42.85 + 11.33\ \chi.$
$sy \cdot x = 10.57,\ r^2 = 0.281.$
t for slope $= 4.64,\ P < 0.001.$

that marked variations in the depth of anesthesia did occur and were difficult to avoid. There is no doubt, however, that, whatever the effect of anesthesia, higher flows will permit greater oxygen uptake by the experimental animal and this effect is a linear relationship in the range studied.

The data in Fig. 1 were compiled by describing 120 separate observations on 57 dogs. It is of interest to document the effect of flow changes in a single animal. This was done in Figs. 2 and 3 where it is noted that changes in oxygen consumption will follow changes in flow regardless of the time relationship between high and low flows.

Venous oxygen saturation increases in linear fashion as flow increases in the range studied as noted in Fig. 4. In this large number of observations, although there is much scatter, venous saturation is more likely to be 70 per cent or above at flows of 2.4 L. per square meter of body surface or above.

That the rise in oxygen consumption with increases in flow is not related to changes in perfusion pressure is demonstrated in Fig. 5 in which there is no apparent relationship between flow and mean arterial perfusion pressure.

TABLE I. MEAN PHYSIOLOGIC VALUES AND STANDARD DEVIATIONS IN 15 CONSECUTIVE PATIENTS UNDERGOING TOTAL CARDIOPULMONARY BYPASS*

| | |
|---|---|
| Mean arterial pressure | 73 mm./Hg ± 19 |
| Mean venous pressure | 11 mm./Hg ± 8 |
| Arterial oxygen saturation | 99% ± 6 |
| Venous oxygen saturation | 70% ± 7 |
| Fall in pH* | -0.05 limits ± 0.1 |
| Fall in buffer base* | -2.03 mEq. limits ± 3 mEq. |
| Partial pressure of carbon dioxide | 35 mm./Hg ± 5 |
| Oxygen consumption | 133 c.c./M.²/min. ± 36 |
| Flow | 2.735 L./M.²/min. ± 0.4 |

*Patient described in Table V excluded.

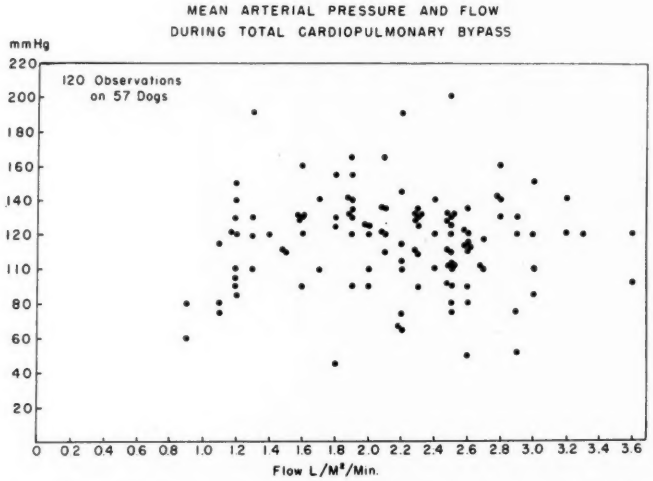


Fig. 5.—This figure demonstrates the lack of correlation between arterial pressure and flow during cardiopulmonary bypass in the dogs studied.

$\hat{y} = 125.15 - 4.32\ \chi.$
$sy \cdot x = 29.28,\ r^2 = 0.011.$
t for slope = 0.79, $P > 0.25$.

*Part II. Human Beings.—*

In the fifteen consecutive human perfusions studied, the mean flow was 2.7 L. per square meter of body surface per minute with a range of 1.74 to 3.3. The mean values for mean arterial pressure, mean venous pressure, arterial saturation, venous saturation, fall in pH, fall in buffer base, and oxygen consumption are shown in Table I. The range of flows utilized in this series resulted in no essential deviation from the normal as seen by these mean values, except for 3 patients to be discussed in detail. In Table II, the venous saturations and oxygen consumptions at the 5-minute, 25-minute and 45-minute mark during perfusions are listed for each patient, along with the corresponding flow which was held constant throughout the entire perfusion period. The oxygen consumption is expressed as a percentage of the predicted basal oxygen consumption from the tables of Clark.[4]

TABLE II. VENOUS OXYGEN SATURATION AND OXYGEN CONSUMPTION RELATED TO BASAL PREDICTED OXYGEN CONSUMPTION DURING TOTAL CARDIOPULMONARY BYPASS AT HIGH FLOWS IN 15 CONSECUTIVE PATIENTS*

| | | 5 MINUTES | | | | 25 MINUTES | | | | 45 MINUTES | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PATIENT | FLOW (L./M.²/MIN.) | VENOUS $O_2$ SAT. (%) | $O_2$ CONSUMPTION (C.C./M.²/MIN.) | PREDICTED $O_2$ CONSUMPTION (C.C./M.²/MIN.) | % PREDICTED $O_2$ CONSUMPTION | VENOUS $O_2$ SAT. (%) | $O_2$ CONSUMPTION (C.C./M.²/MIN.) | % PREDICTED $O_2$ CONSUMPTION | VENOUS $O_2$ SAT. (%) | $O_2$ CONSUMPTION (C.C./M.²/MIN.) | % PREDICTED $O_2$ CONSUMPTION |
| 1 | 2.74 | 71 | 98 | 129 | 76 | 59 | 120 | 93 | 51 | 156 | 120 |
| 2 | 2.86 | 69 | 127 | 147 | 86 | 67 | 181 | 123 | 53 | 190 | 129 |
| 3 | 2.73 | 76 | 120 | 153 | 79 | 71 | 127 | 83 | 58 | 177 | 106 |
| 4 | 2.54 | 74 | 112 | 166 | 68 | 70 | 124 | 76 | | | |
| 5 | 3.05 | 68 | 121 | 144 | 84 | 66 | 154 | 107 | 61 | 177 | 122 |
| 6 | 3.52 | 75 | 90 | 162 | 56 | 69 | 153 | 95 | 74 | 185 | 114 |
| 7 | 2.78 | 77 | 106 | 152 | 69 | 79 | 101 | 66 | | | |
| 8 | 3.00 | 68 | 171 | 173 | 99 | | | | | | |
| 9 | 2.58 | 72 | 129 | 163 | 78 | | | | | | |
| 11 | 1.74 | 75 | 83 | 133 | 63 | 81 | 79 | 60 | 72 | 90 | 68 |
| 12 | 2.64 | 79 | 73 | 177 | 60 | 75 | 87 | 71 | 79 | 89 | 73 |
| 13 | 2.68 | 82 | 113 | 149 | 75 | 74 | 121 | 81 | | | |
| 14 | 2.47 | 72 | 96 | 174 | 50 | 63 | 156 | 90 | | | |
| 15 | 3.30 | 70 | 184 | 170 | 108 | 63 | 264 | 156 | | | |

*Patient described in Table V excluded.

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It is of great interest that the oxygen consumption varied from 50 to 156 per cent of the predicted consumption from patient to patient, and also varied greatly in different samplings from the same patient, with a distinct tendency toward higher oxygen consumption in the latter periods of perfusion. Thus, of seven patients sampled at the 45-minute mark, 5 had oxygen consumptions over 100 per cent of the predicted value. Since the predicted value is based on observations in unanesthetized patients and it is known that anesthesia alone depresses oxygen consumption, the high values of oxygen consumption are surprising under the conditions of cardiopulmonary bypass. These findings would tend to invalidate a rigid method of calculating an optimal flow rate entirely on the basis of predicted basal oxygen consumption, or predicted basal cardiac output. The patient can have requirements well above basal levels under conditions of cardiopulmonary bypass.

Venous oxygen saturations below 70 per cent occurred only in those patients whose oxygen consumption was 86 per cent or above of the predicted basal consumption. However, in a small number of patients, perfused at 3 L. per square meter per minute or above, oxygen consumption above basal levels was supported during extracorporeal circulation with venous oxygen saturations of 70 per cent or more. This flow range, therefore, will maintain more normal findings in the venous saturation when oxygen consumption is above basal levels than will lower flows more closely approximating cardiac output under anesthesia.

Despite an attempt to avoid metabolic acidosis in all patients by utilizing high flows, there were three instances in this series of 15 patients in whom there was significant deviation from the normal values in pH and whole blood buffer base. The patient whose studies are shown in Table III had a low

TABLE III. THE EFFECT OF ARTERIAL UNSATURATION IN THE PRODUCTION OF MILD METABOLIC ACIDOSIS AT HIGH FLOWS

| PATIENT NO. 1—FLOW 2.74 L./M.²/MIN. PREDICTED OXYGEN CONSUMPTION—129 C.C./M.²/MIN.* | | | | | |
|---|---|---|---|---|---|
| | *Control* | *5 min.* | *25 min.* | *45 min.* | *15 min. post-perfusion* |
| pH | 7.37 | 7.34 | 7.30 | 7.26 | 7.21 |
| Buffer base, mEq./L. | 42 | 41 | 40 | 39 | 38 |
| Partial pressure of carbon dioxide, mm. Hg | 38 | 38 | 39 | 41 | 44 |
| Arterial oxygen saturation | 102% | 96% | 86% | 81% | 101% |
| Venous oxygen saturation | | 72% | 59% | 51% | |
| Oxygen consumption, c.c./M.²/min. | | 99 | 121 | 157 | |
| Per cent of predicted oxygen consumption | | 76 | 93 | 120 | |

*From tables of L. C. Clark.

arterial saturation which was unsuspected during cardiac bypass because of a faulty cuvette oximeter. Despite what has been shown to be an adequate flow in the remainder of the series of patients, this patient developed mild metabolic acidosis.

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The patient whose studies are shown in Table IV demonstrates that, when there is marked skeletal muscle activity during perfusion, what would ordinarily be an adequate rate will result in low venous oxygen saturations and mild metabolic acidosis. This patient was extremely light during perfusion with excessive motion of the diaphragm and extremities and a very high oxygen consumption. This, then, is a paradoxical situation in which a high oxygen consumption, well above basal levels, is associated with mild metabolic acidosis.

TABLE IV. THE EFFECT OF MUSCULAR EXERCISE IN THE PRODUCTION OF MILD METABOLIC ACIDOSIS AT HIGH FLOWS

PATIENT NO. 2—FLOW 2.86 L./M.²/MIN.
PREDICTED OXYGEN CONSUMPTION—147 C.C./M.²/MIN.*

| | *Control* | *12 min.* | *26 min.* | *46 min.* | *22 min. post-perfusion* |
|---|---|---|---|---|---|
| pH | 7.46 | 7.34 | 7.31 | 7.26 | 7.37 |
| Buffer base, mEq./L. | 42 | 42 | 40 | 40 | 40 |
| Partial pressure of carbon dioxide, mm. Hg | 31 | 37 | 39 | 44 | 31 |
| Arterial oxygen saturation | 104% | 99% | 98% | 94% | 95% |
| Venous oxygen saturation | | 69% | 67% | 53% | |
| Oxygen consumption, c.c./M.²/min. | | 127 | 181 | 190 | |
| Per cent of predicted oxygen consumption | | 86% | 123% | 129% | |

*From tables of L. C. Clark.

The patient whose studies are shown in Table V suffered from primary pulmonary hypertension with no septal defect found at the time of ventriculotomy. Cardiac bypass could not be discontinued on this patient without resulting in acute heart failure, so that the patient was perfused for 8½ hours. A flow rate, which ordinarily would be adequate for the usual duration of perfusion, was demonstrated in this instance to be inadequate in preventing severe metabolic acidosis from occurring after many hours of cardiac bypass.

## DISCUSSION

The relationship between flow and oxygen consumption during cardiopulmonary bypass is an important one. Paneth has stated that as flow is increased during total cardiopulmonary bypass in dogs with a bubble oxygenator from low levels to 1.2 L. per square meter of body surface per minute oxygen consumption increases, and that a further increase in flow above this level does not result in a further increase in oxygen consumption. It is then inferred that flows above this level are unnecessary and perhaps undesirable since trauma to the blood in the extracorporeal circuit is roughly proportional to flow rate.[5]

Anderson and Senning,[6] on the other hand, have reported that increases in flow in the dog during cardiopulmonary bypass with the Crafoord-Senning pump oxygenator results in increasing oxygen consumption until flows above 100 c.c. per kilogram are obtained. At this flow, oxygen consumption during bypass approaches the pre-perfusion control level.

TABLE V. THE EFFECT OF A PROLONGED PERFUSION IN THE PRODUCTION OF MILD METABOLIC ACIDOSIS AT HIGH FLOWS

PATIENT NO. 10—FLOW 2.35 L./M.²/MIN.
PREDICTED OXYGEN CONSUMPTION—169 C.C./M.²/MIN.

| | *Control* | *3 min.* | *23 min.* | *43 min.* | *1 hr.* | *1 hr. 23 min.* | *1 hr. 56 min.* | *2 hr. 18 min.* | *2 hr. 41 min.* | *3 hr. 44 min.* | *4 hr. 43 min.* | *5 hr. 43 min.* | *6 hr. 43 min.* | *7 hr. 43 min.* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| pH | 7.37 | 7.37 | 7.37 | 7.40 | 7.41 | 7.41 | 7.33 | 7.28 | 7.18 | 7.09 | 7.19 | 7.18 | 7.15 | 7.12 |
| Buffer base | 44 | 42 | 41 | 42 | 41 | 39 | 37 | 34 | 30 | 28 | 32 | 33 | 33 | 33 |
| $pCO_2$ | 38 | 32 | 32 | 30 | 28 | 26 | 27 | 25 | 28 | 36 | 31 | 36 | 42 | 46 |
| Venous oxygen saturation | | 67 | 68 | 74 | 63 | 78 | 77 | 77 | 72 | 64 | 70 | 60 | 67 | 49 |
| Oxygen consumption | | 141 | 136 | 119 | 165 | 90 | 104 | 94 | 106 | 138 | 113 | 160 | 163 | 139 |
| Per cent predicted $O_2$ consumption | | 83 | 80 | 70 | 98 | 53 | 61 | 56 | 64 | 82 | 67 | 94 | 97 | 82 |

The purpose of the present study was to obtain sufficient data on oxygen consumption in dogs, using the same apparatus and team as was planned for cardiopulmonary bypass in human beings and, on the basis of these studies, to select the optimal flow range for initial clinical work. An attempt was made to achieve uniformity in all respects except flow rate. Oversaturation of the arterial blood was carefully avoided by the use of a Clark oxygen sensitive electrode. Disc speed was regulated to maintain a partial pressure of oxygen in the arterial blood between 100 and 200 mm. Hg. The results of these laboratory experiments are not in agreement with those recorded by Dr. Paneth and indicate that, at flows above 1.2 L. per square meter of body surface per minute, oxygen consumption continues to rise, as flow was increased, in a linear fashion that is statistically significant. Oxygen availability and, hence, oxygen consumption appear to be depressed at flows significantly lower than normal cardiac output under conditions of anesthesia and normothermia. The reasons for the differences in experimental results are not immediately apparent but it is possible that the bubble oxygenator results in abnormally high $pO_2$ and, hence, because of a large amount of dissolved oxygen in the blood, flow requirements might be less than the requirement when the partial pressure of oxygen in blood leaving the oxygenator is carefully controlled and kept within the normal range.

As a result of these studies, it was elected to perfuse at flows approximating resting cardiac output in the first 15 consecutive clinical cases. The data obtained from these patients indicate the advantages of such a policy. Severe metabolic acidosis was not encountered, awakening from anesthesia was prompt, and all patients left the operating room in good condition, except one who had primary pulmonary hypertension without a septal defect and could not be taken off the cardiac bypass. Oxygen consumption was quite high in almost all patients, with an average consumption approximating the basal oxygen consumption. In those patients with oxygen demands well above the predicted value, the high flow permitted an oxygen consumption above normal without undue widening of the arterial-venous difference.

A surprising finding in one patient was that oxygen consumption above normal basal consumption may be inadequate in preventing mild metabolic acidosis when oxygen demands are excessive and pump output is limited to a flow of resting cardiac output. Under these circumstances, flow may be a limiting factor in oxygen consumption, and, hence, the perfusion defective in meeting body needs.

## SUMMARY AND CONCLUSIONS

The relationship between flow and oxygen consumption has been studied in dogs and the flow range now considered adequate for human cardiopulmonary bypass. Oxygen consumption has been found to increase in a linear fashion as flow is increased in the range over 1.2 L. per square meter of body surface per minute. Venous oxygen saturation increases in a linear fashion as flow is increased, and values of 70 per cent or more are more likely at flows of 2.4 L. per

square meter of body surface, or above, rather than at lower flows. Total body perfusion in human beings, at flows approximating basal cardiac output, will result in no tendency to metabolic acidosis except in three circumstances: (1) a low arterial saturation due to faulty oxygenator function, (2) excessive skeletal muscle activity during bypass, and (3) a prolonged perfusion. The concept of an ideal flow rate as one which is not in itself a limiting factor to oxygen consumption is put forward. This implies that the ideal flow will not bear a constant relationship to body weight or surface area but would be dependent upon oxygen needs as determined by body temperature, muscular activity, and perhaps other unknown factors.

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# PREVENTION OF SURGICAL HEART BLOCK BY THE USE OF SUPRAVITAL STAIN

*Peter Allen, F.R.C.S.(C), J. J. Lederman, B.A., LL.B., and G. J. Pearl, B.Sc., Vancouver, British Columbia*

ONE of the serious complications of open heart surgery, using total cardiopulmonary bypass, is complete heart block resulting from direct surgical trauma to the cardiac conduction system. Surgically induced atrioventricular dissociation occurs most frequently during repair of superiorly and posteriorly placed ventricular septal defects as well as in atrial septal defects of the ostium primum type. In addition, patients with cyanotic congenital cardiac malformations and those with low cardiac reserve are prone to develop atrioventricular dissociation under the stimulus of surgery alone, without actual trauma to the conduction system.[1] Thus, while complete heart block can occur from other causes, direct surgical injury to the conduction system has now become the commonest form.

The anatomy of the conduction system in relation to the right side of the heart is the key to the problem of surgical heart block.[2] The atrioventricular node lies immediately inferomedial to the mouth of the coronary sinus in the right atrium. The bundle of His passes posterior to the annulus of the tricuspid valve and enters the right ventricle adjacent to the infero- or superoposterior rim of the membranous ventricular septum. The common bundle then divides into a right bundle branch which passes distally along the posterior margin of the membranous septum. The left bundle branch pierces the ventricular septum and lies subendocardially immediately inferior to the aortic valve, where it then fans out along the left ventricular aspect of the septum. Variations to this septal anatomic pattern occur chiefly in the presence of a ventricular septal defect of the membranous and posterior types. If the common bundle passes distally along the posterior rim of a ventricular septal defect and divides into the right and left bundle at the *inferior* margin of the defect, there is a greater chance of developing complete surgical heart block than if the common bundle divides at the superoposterior margin of the ventricular septal defect and only the right bundle passes inferiorly along the posterior margin of the defect. At present, during corrective cardiac surgery, there is no way of knowing which anatomic type is present.

From the Department of Surgery, University of British Columbia, Vancouver, B. C., Canada, and British Columbia Medical Research Institute.

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We have been able to avoid surgical heart block by continuous monitoring of the electrocardiogram during the repair of ventricular septal or ostium primum defects. As dissociation changes in the electrocardiogram are observed, the surgeon is warned of his proximity to the conduction tissue. In this situation, by altering the suture placement, we have avoided complete and possibly fatal heart block on several occasions. However, if one induces potassium arrest, the heart is electrically inactive and the safeguard of a continuous electrocardiogram is lost. Indeed, some surgical centers have experienced an increased incidence of third degree heart block when cardioplegic agents have been used, the inference being that the bundle of His is more easily damaged when surgeons are unable to observe gross or electrical cardiac activity.[3]

To substitute for the loss of electrical activity in potassium arrest, we have attempted to visualize the conduction system by developing a supravital stain which (a) is transitory in action, (b) does not destroy the conduction tissue, and (c) is nontoxic in the general circulation.

## METHODS AND RESULTS

By trial and error a solution of elemental iodine was selected as the ideal supravital stain and tested in the hearts of dogs recently killed in other experiments. Five per cent elemental iodine dissolved in 10 per cent sodium iodide was the most satisfactory stain used to visualize the cardiac conduction tissue, clear visualization lasted 3 to 5 minutes before fading. Weaker solutions did not give a constant staining reaction and occasionally no staining occurred until the 5 per cent iodine solution was used. Stronger solutions, up to 10 per cent elemental iodine in 10 per cent sodium, gave more intense visualization for longer periods which were considered unnecessary. Due to the depressing effect of potassium on cardiac function, sodium (Na I) was used as the vehicle for dissolving the 5 per cent elemental iodine.

Staining reactions were repeated in the hearts of live dogs. Eight mongrel dogs were given a Pentothal Anectine anesthetic and placed on the bubble oxygenator with total cardiopulmonary bypass, following which potassium arrest was instituted using the Melrose technique.[4] The right ventricle was opened and the conduction tissue stained with varying solutions of elemental iodine in 10 per cent sodium iodide. The intensity and duration of the staining reaction was recorded. Potassium arrest was terminated after 10 minutes and, following return of sinus rhythm, the pump-oxygenator was discontinued.

In 7 of the 8 dogs, good staining visualization of the bundle of His and, particularly, the right bundle branch was obtained (Fig. 1). Of the seven successfully stained hearts, six were quickly restored to sinus rhythm at the completion of 10 minutes of induced cardiac arrest, while one dog developed uncontrolled ventricular fibrillation as the result of faulty perfusion technique. In one dog, the bundle failed to stain due to the constant wash

of blood across the endocardium from several sources uncontrolled by suction. The endocardium overlying the conduction tissue must be dry before staining can occur, indeed, blood from any source will prevent visualization of the common bundle or its branches by the iodine method.

In the 8 dogs, staining was commenced with 0.5 per cent elemental iodine followed by 1 per cent, 2 per cent, 4 per cent, and 5 per cent solutions. The 0.5 per cent to 4 per cent solutions gave inconstant visual reactions but 5 per cent solutions stained clearly for several minutes before fading. In a

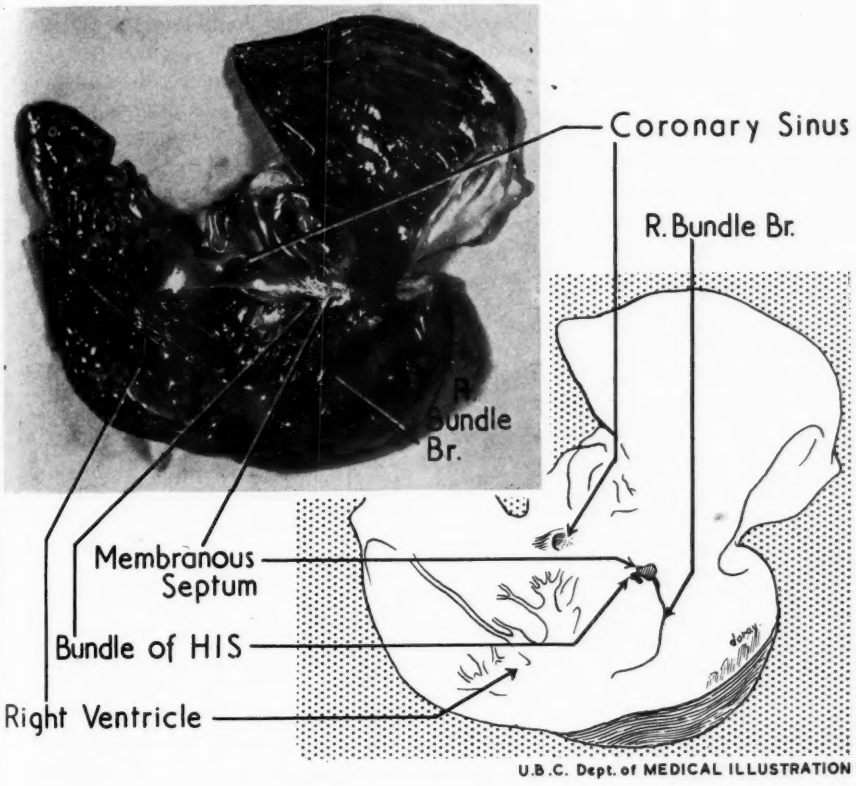


Fig. 1.

series of 4 dogs, 10 per cent iodine solution stained the conduction tissue intensely for 7 to 15 minutes which was longer than necessary. Although sinus rhythm was restored in the 4 cases, the period of resuscitation, after 10 minutes of potassium arrest, was distinctly prolonged and more difficult when compared to the cases using 5 per cent iodine. It is suggested that the more intense staining reactions (10 per cent) are dangerous and can produce permanent damage to the conduction system.

General toxicity of the 5 per cent elemental iodine solution was tested by slowly injecting 3 to 5 c.c. into the pulmonary artery of 4 dogs, all of which recovered completely and without incident. Since the amount of 5 per cent iodine solution used for staining is only 1 c.c. there appears to be a wide margin of safety.

Fixed sections of dog myocardium containing the right bundle branch were stained with periodic acid-Schiff (PAS). The muscle and conduction tissue contained pink staining cytoplasm that was attributed to glycogen. To prove this, sections were first immersed in diastase before PAS staining, and no pink cytoplasm was seen in any tissue. Next, histologic sections of myocardium containing the right bundle branch were immersed for periods up to 72 hours in 5 per cent iodine solution in 10 per cent sodium iodide and then stained with PAS. Microscopically, these sections all showed the same amount of pink staining glycogen in the conduction tissue as did the control sections stained only with PAS but not immersed in iodine.

This would suggest that the iodine stain, while combining with glycogen of cardiac conduction tissue, does not destroy it, nor produce irreversible changes during the short periods of contact at surgery.

## DISCUSSION

In the last three years, great advances have been made in the treatment of surgically induced heart block. Initially, intravenous and rectal Isuprel* were used to increase ventricular rate and maintain a satisfactory cardiac output in the presence of complete atrioventricular dissociation. Some patients, however, become refractory to Isuprel[1] and develop ventricular bradycardia with poor coronary artery filling and fatal cardiac arrest. The brilliant development by the Minnesota group of a myocardial wire and mechanical cardiac pacemaker has successfully treated many patients with severe surgical third degree heart block.[5]

Our purpose has been to develop a method of *preventing* surgical heart block by visualizing the bundle of His and, during the short period of supravital staining, to effect safely the placement of closing sutures in the ventricular septal or ostium primum defects. We have used the staining technique on 3 patients. The first was a 9-year-old boy with a common ventricle that was repaired by making an Ivalon septum. It was difficult to determine the exact site of suture placement since no remnant of septal tissue was present. After potassium arrest was instituted, 5 per cent elemental iodine in 10 per cent sodium iodide solution was painted on the endocardium of the common ventricle to show a lacy conduction network on the supero-anterior and superoposterior aspects. In this way, sutures were placed in the wall where conduction tissue was absent, and upon completion of the repair the potassium arrest was terminated with rapid restoration of sinus rhythm.

The second patient was a 3-year-old boy with advanced pulmonary hypertension and a large ventricular septal defect. Following potassium

*Available from Winthrop Laboratories, New York, N. Y.

arrest it was possible to see that the defect involved the entire septum from the crista supraventricularis distally to the pulmonary and aortic valves which were separated by a millimeter of fibrous tissue. Five per cent iodine solution outlined either the common bundle or the right bundle branch in the postero-inferior margin of the septal defect and, as a consequence, sutures were placed wide of this tissue in effecting a repair. After completion of arrest, sinus rhythm was restored.

The third case involved an 8-year-old girl with a membranous ventricular septal defect and a pulmonary artery pressure of 90/40 mm. Hg. Following induced potassium arrest, 5 per cent iodine solution was painted along the margins of the defect but failed to show conduction tissue. Following repair of the defect, the patient developed permanent third degree heart block. It is probable the conduction bundle failed to stain because it was deep within the ventricular septum and beyond the penetration of the iodine solution.

This is a preliminary report, based on experimental work and limited patient application. However, we believe that, with further experience, supravital staining of the conduction system in the right ventricle will add to the safety of potassium arrest and may reduce the incidence of complete heart block in the electrically inactive myocardium.

SUMMARY

The desirability of transitory visualization of the bundle of His in the potassium arrested heart is discussed.

Five per cent elemental iodine in 10 per cent sodium iodide was the most satisfactory supravital stain used to outline the cardiac conduction system of the right ventricle.

Experimental work and brief patient experience is outlined.

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# ACUTE PERICARDIAL FAT NECROSIS

## Report of a Case

*Martin H. Chester, M.D., Gardena, Calif., and*
*John B. Tully, M.D., Hawthorne, Calif.**

THE PERICARDIAL fat necrosis syndrome was first recognized as a clinicopathologic entity by Jackson, Clagett, and McDonald.[1] The authors enphasized the fact that this lesion can simulate the symptomatology and physical findings of acute myocardial infarction, pulmonary embolus, or hemorrhage into a neoplasm of the lung. The roentgenographic appearance may easily be misinterpreted as a primary or metastatic carcinoma of the lung, a localized pleural effusion, pleuropericardial cyst, or mesothelioma. A consideration of this disease entity is, therefore, warranted in any patient with unexplained precordial pain, pleuritic pain in the left lower chest, and an associated juxtacardiac mass.

Jackson, Clagett, and McDonald[1] reported 3 cases of pericardial fat necrosis in which exploratory thoracotomy was performed from 7 to 10 weeks after the acute onset of symptoms. In the following case of pericardial fat necrosis, an exploratory operation was made 13 days after the acute onset of symptoms. This case afforded us an opportunity to observe and describe the pathologic changes of the acute phase of this recently recognized clinical syndrome. To the best of our knowledge a description of the early stage of this lesion has not appeared in the literature.

### CASE REPORT

The patient was a 46-year-old white male carpenter. On Feb. 4, 1958, he was treated for an upper respiratory infection. He had fever, nonproductive cough, generalized aching, and fatigue. A chest roentgenogram was found to be within normal limits (Fig. 1, *A*). The patient recovered within a few days with antibiotic therapy.

On Feb. 14, 1958, the patient experienced the sudden onset of left lower anterior chest pain and cramping epigastric pain. The onset of this pain occurred during a severe lifting effort, when it is estimated that he exerted a lifting force equal to 150 to 200 pounds. This pain radiated to the left shoulder and increased in severity on deep inspiration. The pain, subsequently, became excruciating and caused a brief episode of syncope. He was hospitalized on Feb. 17, 1958, with an admitting diagnosis of acute myocardial infarction. His past medical history revealed that there were recurrent duodenal ulcer symptoms and one episode of bleeding during the past 18 years. However, the patient had been free of ulcer symptoms during the past year.

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*Department of Pathology, Hawthorne Community Hospital.

Physical examination on admission to the hospital revealed a white man, 5 feet 11 inches tall, weighing 157 pounds. He was pale, sweating, and in excruciating chest pain. Respirations were rapid and shallow, blood pressure 98/68 mm. Hg, and pulse 126. He was unable to take a deep breath because of the exacerbation of the chest pain. Auscultation of the chest elicited normal breath sounds and fremitus in the lung fields. An unusual, soft, low-pitched rub was heard during expiration, synchronous with systole at the cardiac apex. This sound closely resembled the sound produced by compressing a wet sponge. It could easily be distinguished from the classical friction rub of a pericarditis. The rub disappeared on the third hospital day.

Although there was moderate improvement of the pain with the administration of Demerol and oxygen, the patient remained in a state of shock. With the administration of Levophed by intravenous drip, his general condition finally improved. Within several hours his blood pressure stabilized to 126/82 mm. Hg, and the Levophed was discontinued.

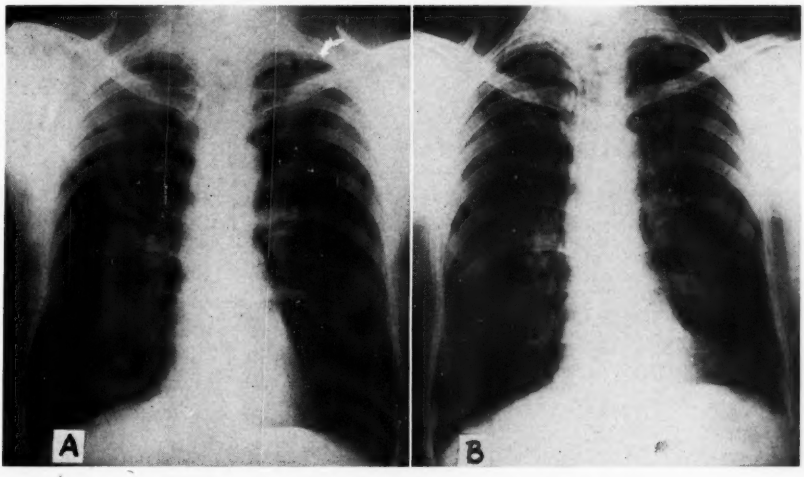


Fig. 1.—*A*, Roentgenogram taken 10 days prior to the onset of symptoms.
*B*, Roentgenogram taken 3 days after the onset of symptoms. Note the appearance of a juxtacardiac mass represented by the area of increased density along the left border of the cardiac shadow.

An electrocardiogram was normal, except for an occasional ventricular presystole. The corrected sedimentation rate was 12 mm. Chest roentgenograms (Figs. 1, *B* and 2) demonstrated a juxtacardiac mass along the anterior aspect of the left cardiac border.

Serial electrocardiograms and serum transaminase determinations were normal. Tomograms of the juxtacardiac mass revealed several ovoid areas of decreased density (Fig. 3).

During the following week the patient's general condition improved considerably, although the left chest pain persisted in severity.

On Feb. 27, 1958, an exploratory operation was performed through a left anterolateral incision in the sixth intercostal space. A firm ovoid mass, measuring 10 by 7 cm., was identified as the pericardial fat pad. It was adherent to the anterior and lateral aspect of the pericardium and the anterior chest wall. There were also adhesions binding the lingula to the pericardial fat pad. The mass was excised by blunt dissection from the pericardium, anterior chest wall attachments, and lingula.

Grossly, the specimen measured 10 by 7.5 by 3.0 cm. (Fig. 4). It consisted of fatty tissue. The external surface was focally indurated and varied from a mottled light brown to a red-black hemorrhagic discoloration. A sharply delineated hemorrhagic area at the

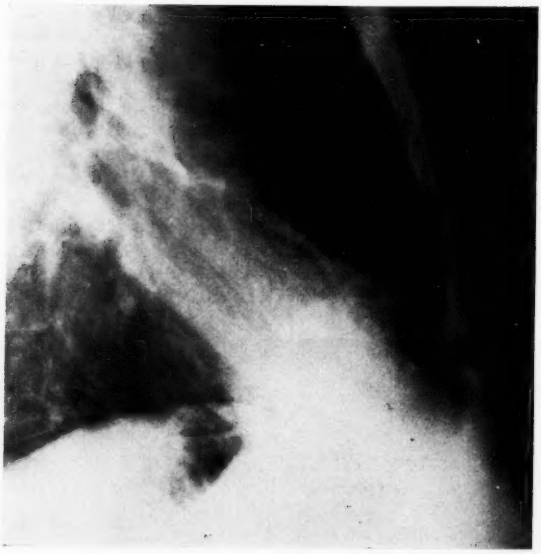

Fig. 2.—Lateral view demonstrating an ovoid density due to the enlarged pericardial fat pad.

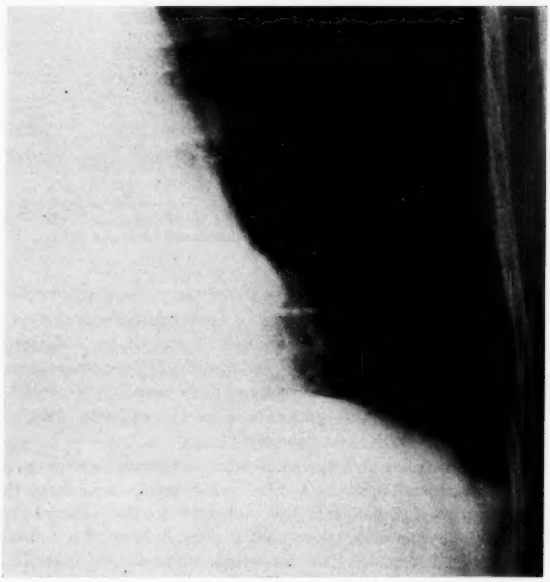

Fig. 3.—Tomographic section study demonstrating saccular radiolucent areas within the pericardial fat pad.

superior end (Fig. 4) measured 2.2 cm. Other areas of subcapsular extravasation of blood were noted. The cut surface a few centimeters from the hemorrhagic area appeared to be homogenous and had a normal fatty color.

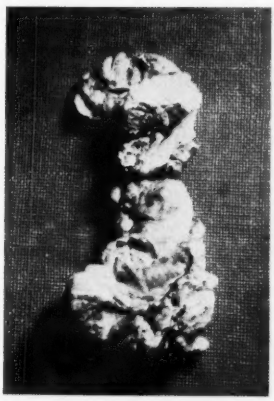

Fig. 4.—Gross specimen of resected pericardial fat. The dark areas contained old blood. The specimen measured 10 by 7.5 by 3.0 cm.

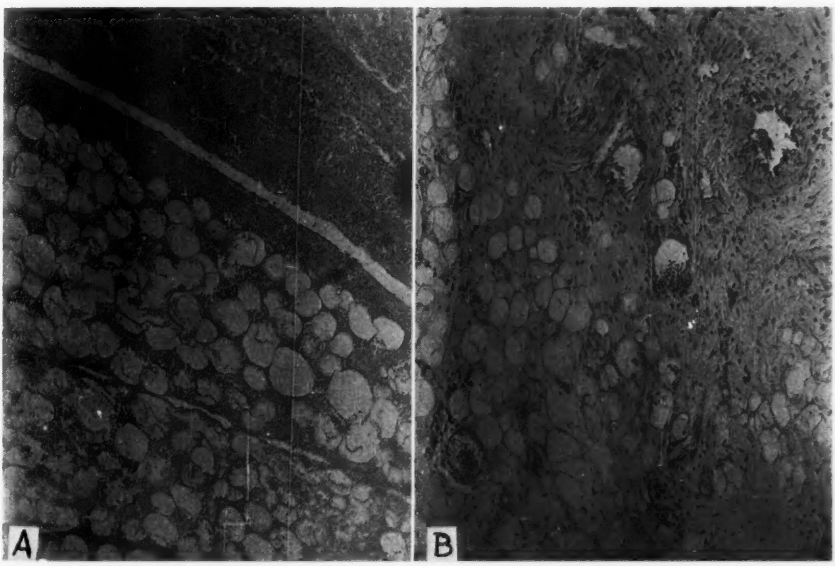


Fig. 5.—*A*, Section of tissue in area of hemorrhagic necrosis showing extravasation of blood and infiltration of inflammatory cells.

*B*, Section of tissue from the periphery of area of hemorrhagic necrosis showing fibrosis and chronic inflammatory cells.

Histologically, the tissue revealed acute hemorrhagic infarction with considerable interstitial hemorrhage, necrosis of blood vessels, and sparse infiltration with polymorphonuclear leukocytes and lymphocytes (Fig. 5, *A*).

At the periphery of the infarcted areas, lipophagic cells were seen and there was a zone of sclerosing granulation tissue infiltrated with acute and chronic inflammatory cells. Within

this tissue, occasional arterioles were seen the walls of which were infiltrated by polymorphonuclear leukocytes (Fig. 5, *B*). These cells spilled over into the perivascular spaces. A few arterioles showed subintimal fibrous thickening of a moderate degree. Bacteria or fungi were not demonstrable.

## COMMENT

The clinical characteristics of this disease, as observed in our patient, were similar to those found in the 3 cases reported by Jackson, Clagett, and McDonald. However, there were several exceptions. This patient was not obese; to the contrary, he was thin and tall. This fact denies the concept that the lesion occurs only in the obese. In addition, there was a temporal and probably a causative relationship between the severe lifting effort and the acute onset of symptoms. The strain of this lifting effort produced the hemodynamic changes and stresses of a Valsalva maneuver. There is strong probability that a traumatic etiology was present in our case. The presence of neurogenic shock and cardiac arrythmia, which were concomitant findings in the early period of hospitalization, caused us to assume that we were treating a case of myocardial infarction with early coronary shock.

Although the pathogenesis of this lesion is still obscure, it is possible that its onset is primarily a "vascular accident" with the resultant extravasation of blood from an artery, hematoma formation and thrombosis. Consequently, the blood supply to the fatty tissue is compromised with the resultant ischemic fatty necrosis.

A comparison of the acute and chronic phases of pericardial fat necrosis would lead one to assume that there is absorption of the extravasated blood, subsequent fibrosis, and ischemic necrosis of fat. This assumption would seem to be justified by observation of the changes in the pericardial fat specimen. The transition from acute hemorrhage to organization, fibrosis, ischemic fat necrosis, and endarteritic changes is clearly demonstrated.

## SUMMARY

1. A case of acute pericardial fat necrosis treated by surgery 13 days after the acute onset of symptoms is presented.

2. The symptoms of acute onset of precordial pain, pleuritic pain in the left chest radiating to the left shoulder, and the demonstration of a juxtacardiac mass in the thoracic roentgenogram are the principal findings in this syndrome. A precordial systolic rub also may be heard.

3. Tomographic section studies may show radiolucent ovoid areas which probably represent normal fat lobules surrounded by hemorrhagic, necrotic, and fibrotic tissue.

4. Histologically, the changes were due to extravasation of blood, ischemic fat necrosis, and fibrosis. Moderate endarteritic changes were also noted.

5. The traumatic etiology is a strong probability in our case.

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# MYOCARDIAL REVASCULARIZATION THROUGH A GASTRIC PEDICLE

## An Experimental Study

*E. Converse Peirce II, M.D., Freeman L. Rawson, M.D., and C. Harwell Dabbs, M.D., Knoxville, Tenn.*

A NUMBER of methods have been and are being used clinically to improve the blood flow to the myocardium in cases of coronary artery disease, and some surgeons are advocating that procedures be done on an almost routine basis as patients approach middle age. The diversity of the methods advocated and the lack of any clear-cut proof that benefit will result put the present clinical work on an uncertain basis. Because of the inherent difficulties of clinical evaluation of coronary disease, compelling proof must be forthcoming from animal experimentation before surgical correction of coronary disease will be a generally accepted procedure. The more promising experimental methods, at present, make use of pedicle flaps of skin or alimentary tract.[1, 7, 10] That such a pedicle flap can carry useful blood to a parenchymal organ has been clearly demonstrated for the kidney.[6] Other procedures are generally theoretically less sound or are too difficult technically. It is likely that intelligent use of pedicle flaps will result in real improvement in coronary artery disease, providing simple production of extracardiac anastomoses will, in fact, lead to greater myocardial blood supply. This preliminary report concerns the formation of an adequate pedicle flap from the stomach and a method for evaluating its efficacy by direct measurement of the myocardial oxygen tension. The stomach was chosen because of its ready accessibility from the chest, its close proximity to the heart, its rich blood supply, and the relative ease of performing plastic surgery on this organ. This method of study was chosen because more usual methods concerned with mortality, or the anatomic demonstration of communications have not yielded clear-cut results.[4, 12]

### MATERIALS AND METHODS

Mongrel dogs are operated on in a standard manner through the left eighth interspace. A portion of the stomach is delivered through an incision in the ligamentous portion of the diaphragm (Fig. 1). A flap about 2½ inches on a side, based on the superior portion of the lesser curvature, is raised on the anterior aspect of the stomach, much as is done in performing a Janeway gastrostomy (Fig. 2). The mucosa is removed from this flap, the stomach is closed in a

From the Surgical Research Laboratory of the East Tennessee Tuberculosis Hospital, and the Departments of Surgery and Medicine, the Acuff Clinic, Knoxville, Tenn.
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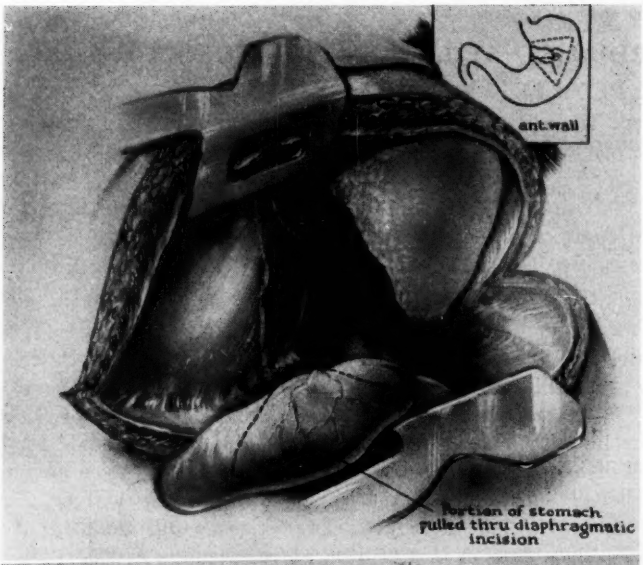
Fig. 1.



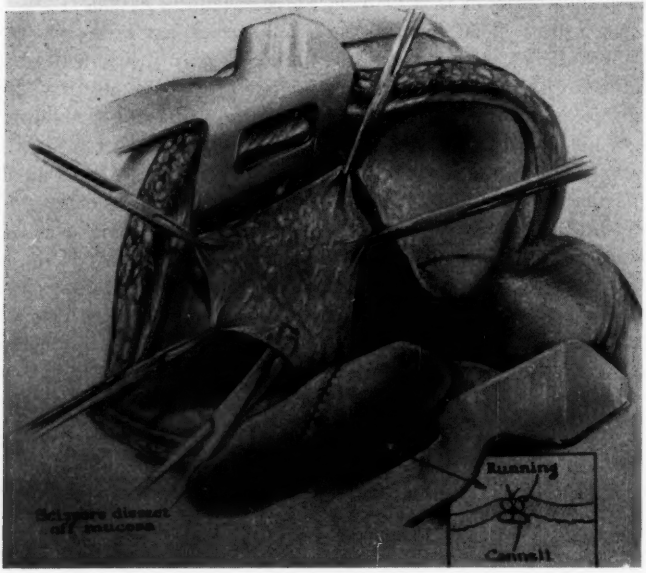




Fig. 2.

Fig. 1.—The stomach is readily accessible through the diaphragm adjacent to the apex of the heart.

Fig. 2.—The plastic flap based on the lesser curvature is freed of mucosa and the stomach is then closed in a routine manner.

Fig. 3.

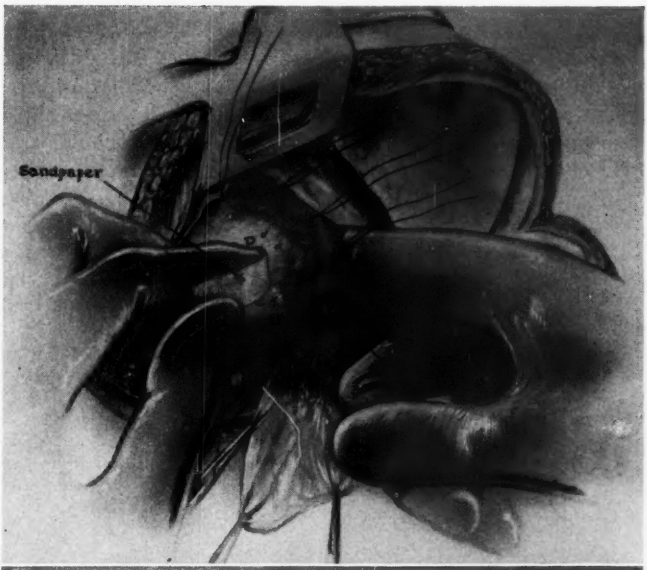



Fig. 4.

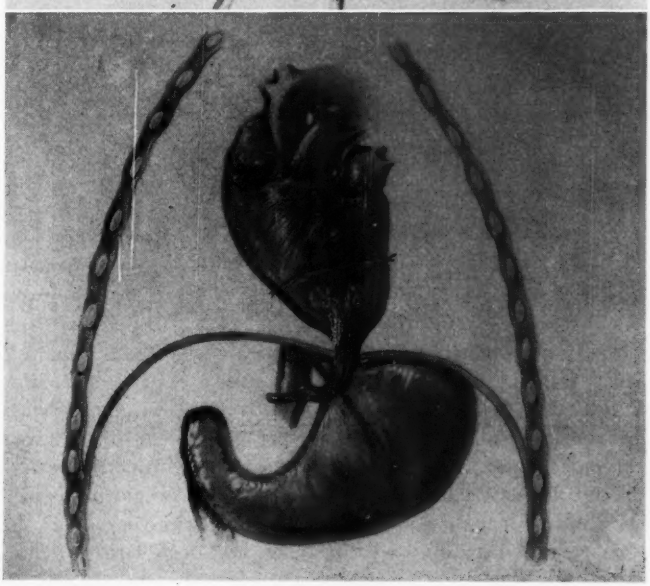


**Fig. 3.**—After the apex of the heart has been abraded with sandpaper, the prepared flap is sutured to the heart with fine silk. The position of the sutures is shown in the figure.

**Fig. 4.**—The general relationships of the cardiac apex, the pedicle flap, the lesser curvature of the stomach, and the blood supply via the left gastric artery are shown.

routine manner, and the diaphragm is closed about the base of the pedicle. A long pericardial incision is made parallel to the phrenic nerve, and the apex of the heart is delivered and abraded lightly with fine "wet-dry" sandpaper (Fig. 3). The raw surface of the pedicle flap is then attached to the myocardium by interrupted sutures of 4-0 silk. As applied, the flap generally measures 8 to 10 cm. in diameter and covers portions of all the major coronary arteries. Closure of the pericardium provides firm pressure to approximate the flap to the myocardium. The chest is then closed in a standard manner, without drainage. The

Fig. 5.—A completed pedicle flap at approximately 6 weeks. There is relatively little pericarditis. The flap is firmly attached to the heart. *RV*—right ventricle; *LV*—left ventricle; *LA*—left atrial appendage; and *F*—pedicle flap. A ligature has been loosely placed about the pedicle of the flap preliminary to occluding its blood supply.

relationship of the completed pedicle to the heart, diaphragm, and stomach is seen in Figs. 4 and 5. The procedure is generally carried out without blood replacement and the animals present no special postoperative problems.

The second thoracotomy for study is generally done 6 to 8 weeks later. Surviving animals are sacrificed. Postmortem arterial injections are done in a few animals and, in most instances, routine histologic preparations are made.

Oxygen studies, mainly of the left ventricular myocardium, have been carried out in control dogs, dogs with pedicles, and dogs with pedicles in whom previous ligation of the anterior descending coronary artery had been done. Ligation of the anterior descending coronary has always been performed at its take-off from the left coronary artery. The platinum microelectrode as described by Davies and Brink[5] and by Montgomery and Horowitz[9] has been used for these studies. A special electrode developed by George Peirce at the University of Pennsylvania has proved useful for myocardial measurements (Fig. 6).[11] In

practice, the platinum microelectrode transmits a current, which can be read on a sensitive galvanometer, that is directly proportional to the oxygen available to it, when 0.6 volt driving force is delivered to the electrode. This appears to be based on the fact that the current is transmitted by the electrolysis of oxygen and, even in complicated solutions, this reaction accounts for the entire flow of current. The electrodes give only relative readings but are sufficiently stable for acute experiments. The changes in oxygen tension are of great magnitude.

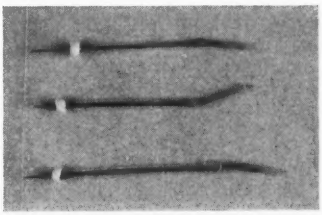


Fig. 6.—Three platinum microelectrodes are shown in approximately natural size. Platinum wire is sealed in glass and the tip is sharpened. The illustrated electrodes have tips of varying length, shown distal to the white bead. A drop of mercury in the tiny polyethylene tube is used to make contact with the lead wire.

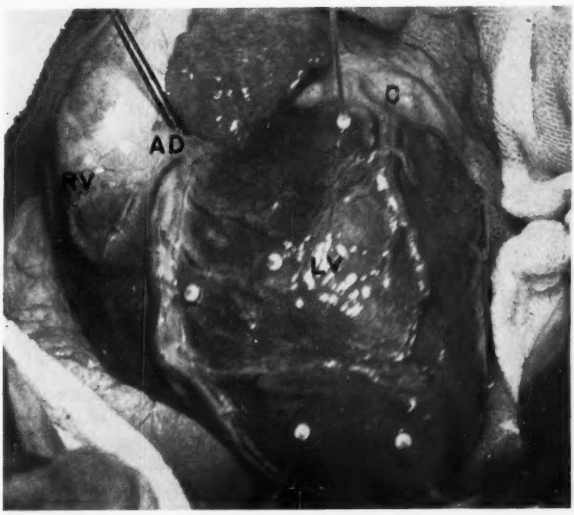



Fig. 7.—Five electrodes are shown in place in a normal beating heart. They can be located where desired in relationship to the various coronary arteries. In animals, with a pedicle flap, usually at least one electrode is placed in the flap itself. *RV*—right ventricle; *LV*—left ventricle; *AD*—anterior descending coronary artery; *C*—circumflex coronary artery. A ligature has been placed about the origin of the anterior descending artery preparatory to ligation.

Multiple electrodes may be used and they may be read continuously or intermittently. For simplicity, five electrodes appropriately spaced are used and each electrode is read at one-minute intervals. In general, some electrodes are placed in the area which would be expected to become ischemic on anterior descending coronary ligation, some are placed in the area supplied by the circumflex artery, and generally one is placed in the flap itself (Fig. 7).

RESULTS

There have been only three deaths in twenty-five preparations. It is necessary to remove the mucosa meticulously because, in 2 dogs, mucosa-lined pockets developed between the flap and the surface of the heart. All of the other dogs had firmly adherent pedicles and were satisfactory for study. The presence of vessels crossing between pedicle and myocardium was demonstrated in the injection preparations and vessels of arteriolar size were frequently seen in routine sections.

Although the number of experiments is much too small to justfy any conclusions, some very provocative data have been obtained. The myocardial electrodes are relatively less sensitive to the dog's breathing 100 per cent oxygen than are similar electrodes placed in the skin. A change from air to oxygen increases the arterial oxygen tension about fivefold and, in the skin, increases of from 300 to 700 per cent in readings are quite usual. In the heart, increases of 50 to 200 per cent are more often the rule (Fig. 8). Electrodes placed beyond

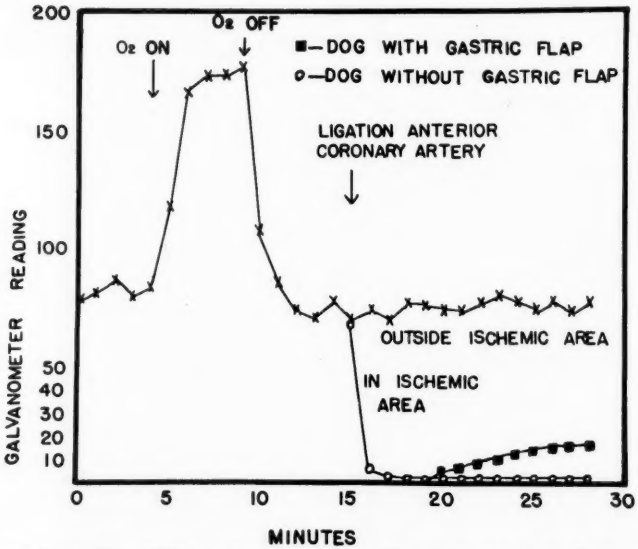



Fig. 8.—Galvanometer readings of several myocardial electrodes have been averaged. The pattern illustrated does not always occur. For explanation of figure see text.

the zone of ischemia produced by coronary artery ligation show relatively constant readings. Electrodes in the zone of ischemia show an extremely rapid drop, essentially to zero. In control dogs, these low readings continue for a matter of minutes until the dog dies of ventricular fibrillation, which has occurred in every instance. In some dogs with flaps, the electrode readings follow this same pattern, but in some there is a gradual, although modest, climb indicating an increase in the oxygen tension in the ischemic area. Subsequent ligation of the pedicle in these dogs again reduces the readings nearly to zero. These studies appear to demonstrate a definite flow of blood through the flap to the heart in at least some of the animal preparations.

A frequent problem has been the sudden onset of ventricular fibrillation, which has terminated many experiments prematurely. This difficulty has not yet been solved.

## DISCUSSION

Clinical evaluation of coronary artery disease is extremely difficult, because of the almost constant psychic overlay in patients and the subjective nature of the symptoms. Objective evaluation by exercise tolerance or by reversion of the electrocardiogram toward normal is not satisfactory since in the first place there is a tendency for angina to improve spontaneously and, in the second place, a revascularization procedure may itself produce abnormal electrocardiographic changes.

The problem is complicated in dogs because of the ease with which blood flow to an individual area of the heart may be increased by development of intrinsic intracoronary anastomoses.[3, 8, 12] In the first place, any procedure, including a sham operation on the heart, will apparently protect a dog to a certain extent from myocardial ischemia following ligation of a coronary artery.[8] The only satisfactory explanation for this is development of a collateral circulation within the heart. In the second place, one cannot produce a preparation with chronic coronary insufficiency by coronary artery ligation.[12] Dogs with all three main coronary trunks ligated in stages may be able to run normally on a treadmill.[3]

It would appear then that the experimental results must be viewed circumspectly. Improvement in mortality in a given experimental preparation following coronary artery ligation, even if statistically valid, does not indicate that the procedure increases the extrinsic blood supply of the heart. It may reflect simply an increase in the effective anastomoses between the existing myocardial vessels. In like manner, the demonstration of vessels passing between the pedicle and the heart anatomically does not indicate the direction or the magnitude of blood flow. The coronary backflow method, advocated by Beck, gives some indirect information regarding the status of both intra- and extracoronary anastomoses. It can be shown, for instance, that, early after a coronary sinus anastomosis, back bleeding from the cut circumflex artery is increased and that the increase is less if the coronary sinus anastomosis is clamped.[8] Since the procedure involves reversing the blood flow and decreasing the pressure in the bed of the circumflex artery, it is not physiologic and interpretation of the data is hazardous.

Measurement of myocardial oxygen tension records objectively and graphically by means of an important physiologic variable what is quite obvious visually following major coronary artery ligation, the appearance of an area of ischemia marked by cyanosis and sometimes by paradoxical motion. Except for the necessity of leaving the chest open, the method is a physiologic one. Changes in potential blood supply resulting from the experimental flap can be elucidated by measuring the oxygen tension change when the flap is clamped. It should be possible with this method to determine if there is a useful increase in extracoronary blood flow from the gastric flap in the present preparation or, for that matter, if a variety of other preparations increase extracardiac flow.

In this regard, it seems particularly unfortunate that methods of treating myocardial insufficiency by procedures such as internal mammary ligation, which defy evaluation, should be used clinically.

Since experiments are frequently interrupted by ventricular fibrillation, it is planned to modify the experimental procedures a little to decrease the incidence of this arrhythmia and permit oxygen tension data to be obtained over longer periods of time. Contemplated modifications include: (1) slow coronary occlusion with Berman "buttons,"[2] followed by oxygen tension studies on ligation of the pedicle, (2) surgical implantation of microelectrodes, so that studies can be done in closed chest preparations, and (3) ligation of smaller branches than the anterior descending coronary artery to decrease the incidence of ventricular fibrillation.

SUMMARY

A satisfactory method of fashioning a vascular pedicle from the stomach and anastomosing it to the apex of the heart is described. The evaluation of blood flow in the pedicle by measurement of myocardial oxygen tension is discussed. No conclusions are drawn concerning the efficacy of the experimental procedure in increasing the myocardial blood supply in this preliminary report. Plans for improved experimental methods are given.

ADDENDUM

One patient has had the described operation for intractable angina pectoris. Technically, the operation was entirely satisfactory and there were no digestive or diaphragmatic problems postoperatively.

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# FORWARD VERSUS RETROGRADE CORONARY PERFUSION FOR DIRECT VISION SURGERY OF ACQUIRED AORTIC VALVULAR DISEASE

*Norman E. Shumway, M.D., San Francisco, Calif.*

THE aortic valve is the most impregnable orifice of the human body. It may well be the last anatomic frontier of surgery. Its diseases are among the most difficult cardiac lesions to treat. Moreover, the risk of attempting to modify the pathologic architecture of the aortic valve often assumes dimensions all out of proportion to the amount of relief usually derived from a "successful" procedure. Many heart surgeons refuse to deal with any deformity of the aortic valve, and most are skeptical of the efficacy of surgery in calcific acquired disease and congenital stenosis without poststenotic dilatation. Until the mortality now associated with excursions in front of the aortic valve is substantially decreased, pleas for early operation are likely to continue unheard by cardiologists. This report will deal primarily with experimental efforts to provide a smoother avenue to the aortic tunnel.

Human application of Alexis Carrel's experimental work on the aortic and pulmonary valves, reported in 1910[1] and 1914[2], came slowly and at great cost. Blind approaches, initiated by Tuffier,[3] Brock,[4] Smithy and Parker,[5] and Niedner[6] and popularized by Bailey[7] and Glover[8] and their associates, are clearly inferior to any direct method the performance of which is attended by a respectable operative risk. After the work of Neville and Clowes[9] exposed Cookson's[10] misconception that the sucking action of the beating heart would predispose to coronary air embolism, Lewis,[11] Swan,[12] and Lillehei[13] operated upon the aortic valve under direct vision. The open method achieved introduction if not approbation.

In the 3-year period since the inauguration of direct vision surgery for the aortic valve, certain improvements and changes have developed. The always hurried, often frantic, valvulotomy, during hypothermia alone, is obsolete. Lewis[14] prefers the membrane oxygenator and moderate hypothermia with coronary perfusion; this permits unhurried, calculated surgery of the aortic valve. In most cases, Lillehei[15] feels that the bubble oxygenator with some kind of coronary perfusion at normal temperature is the least difficult technique.

From the Department of Surgery, Stanford University School of Medicine, Stanford University Hospitals, San Francisco 15, Calif.

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It seems at this writing that congenital aortic stenosis differs importantly from acquired disease in that an oxygen source is not essential for the myocardium during valvuloplasty. Depleted cardiac reserve, usually present in acquired lesions, necessitates the use of some kind of coronary perfusion. Elective cardiac arrest with potassium citrate, acetylcholine, ventricular fibrillation, or anoxia is probably not advisable in most cases of acquired aortic disease.

The experiments reported herein pertain to the delineation of improved methods for coronary perfusion during exposure of the aortic orifice. Cardiopulmonary bypass was achieved by means of a rotating disc oxygenator. It is clear that any method of coronary perfusion suitable for open surgical treatment of aortic stenosis would be equally applicable in the direct attack on regurgitation. Actually, coronary ostial cannulation is more easily accomplished in the dilated aortic ring than in patients with aortic stenosis. All combinations of stenosis and insufficiency may be encountered; in fact, the need for time to correct postvalvulotomy regurgitation was a prime factor in the trend away from hypothermia.

### RETROGRADE CORONARY PERFUSION

Accepting the premise that some kind of coronary perfusion was essential, we turned first to retrograde perfusion of the coronary sinus. Lillehei and his associates[16] are properly credited with the first clinical trial of retroperfusion to achieve direct view of the aortic valve. Initially, the patulous coronary sinus was cannulated with a plastic catheter which was then sutured into position to prevent dislocation during the operative procedure on the aortic valve. The most attractive feature of retrograde perfusion is that the coronary circulation, though reversed, is never interrupted since the necessary cannulation is effected via atriotomy during cardiac perfusion from the heart-lung apparatus. Blood flows from a reservoir into the coronary sinus as soon as the aorta is occluded and incised distal to the valve. Distance from the reservoir to the heart is usually held at 50 to 75 cm. The reservoir is transfused from the arterial limb of the heart-lung machine so that warm, oxygenated blood is always available. Some recent work by Berne, Jones, and Cross[17] suggests that cold blood may be acceptable, but our experience with this is accidental, when blood approached room temperature due to early filling of the reservoir, and no conclusions can be drawn.

Proximity of the atrioventricular node to the coronary sinus warns against suture fixation of whatever cannula is used for the perfusion. Clinically, gross and persistent conduction aberrations have resulted from such a maneuver. The logical alternative to suture-fixation is use of a No. 10 to No. 18 Fr. Foley catheter with a 5 c.c. balloon. Inflation of the balloon with blood or saline solution prevents escape of the catheter from its wedge position in the coronary sinus. The anterior coronary and Thebesian veins are available to transport blood into the cardiac chambers after the Foley balloon is distended but before the onset of retrograde coronary perfusion. This technique was used in

numerous dog experiments with consistent and interesting results. The flow rate for each retroperfusion was between 35 and 55 c.c. per minute. The method is depicted in Fig. 1.

Although animal survival was the rule, certain disadvantages appeared inseparable from the method. A considerable amount of arterialized blood escaped into the right atrium and right ventricle, failing to complete the route through the capillary bed. This fraction of the perfusate was totally denied to the myocardium. Another clear disadvantage was the very small volume of blood emanating from the right coronary ostium. This indicated the left side of the heart was perfused to the near exclusion of the right. Gregg[18] would have

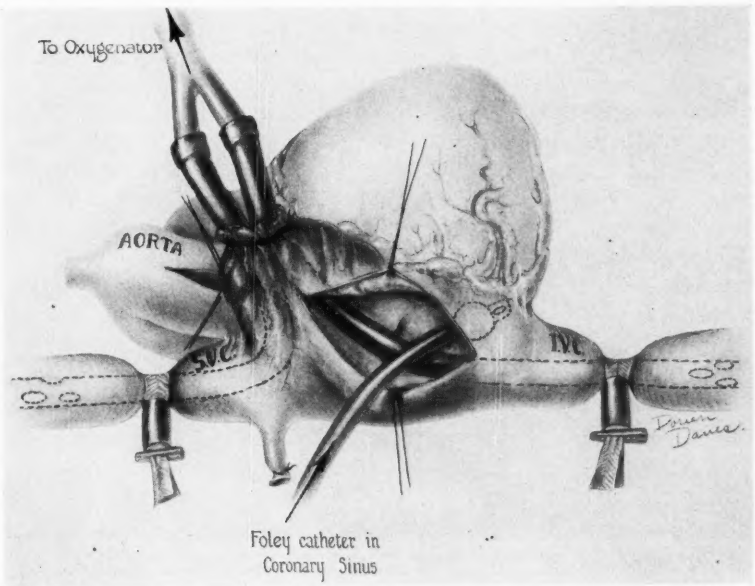


Fig. 1.—An acceptable method for retrograde coronary perfusion.

predicted this phenomenon from his exhaustive studies of the coronary circulation. The third and most striking disadvantage lay in the behavior of the heartbeat during retrograde coronary perfusion. The heart would approach standstill; then, as though the heart were shifting gears, the rhythm returned to the modulated vigor characteristic of the heart during cardiopulmonary bypass. Color of the myocardium remained pink in most experiments. In one animal, complete heart block preceded the routine episode of severe bradycardia. This borderline standstill developed in each animal just after reversal of the coronary circulation.

Postmortem studies in 2 animals were sufficient to strengthen the impression that something other than retrograde coronary perfusion was the answer to the general problem. A huge ulcer and associated thrombus congruent with

the site of the Foley balloon obstructed completely the coronary sinus—right atrial junction in one dog. In the other, similar but less striking changes were observed. The possibility that acute and total occlusion of the coronary sinus may not be serious detracted little from our disappointment in noting that balloon fixation was not unattended by complications.

Duration of retroperfusion was 15 minutes. The perfusate was aspirated and discarded. Flow rates for the body perfusion varied from 65 to 85 c.c. per kilogram per minute.

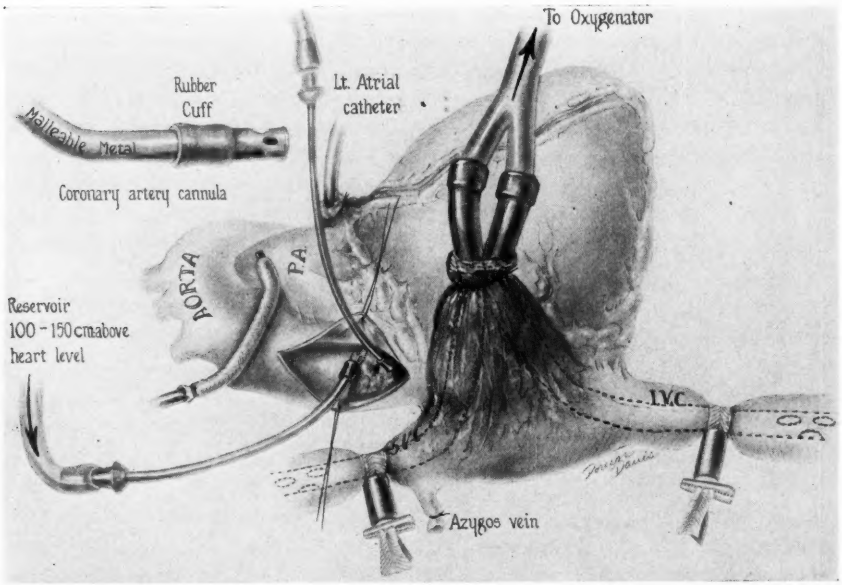


Fig. 2.—Individual cannulation of the coronary ostia. Inset shows detail of cannula.

CORONARY OSTIAL PERFUSION

In turning from retrograde to forward coronary perfusion, the first problem was selection of an appropriate catheter. Studies of the anatomy of the coronary ostia in dogs revealed that in most animals the right coronary ostium was very small, essentially precluding direct cannulation. The left coronary artery frequently trifurcates immediately below the aortic surface thereby preventing any considerable length of cannulation. The catheter, therefore, could not be wedged into the coronary artery, and the need for constant control of the catheter tip indicated that some relatively inflexible material would best meet this requirement. The matter of catheter type suddenly became an important consideration. The design of the malleable metal catheter ultimately evolved is seen in Fig. 2. The rubber cuff prevents reflux of arterialized blood into the operative area, and extra holes in the catheter tip tend to avert obstruction due to changing

angulation of the instrument. Another advantage to malleable metal is the fact that cannulation is accomplished almost instantaneously with visualization of the ostia. No additional forceps is needed to manipulate the cannula into the coronary vessel. The catheter is coated with silicone as are all metal, glass, and rubber surfaces in the extracorporeal heart-lung circuit.

Gravity flow over a distance of 100 to 200 cm. suffices to propel blood from a reservoir into the artery. The flow rate varied from 25 to 50 c.c. per minute, and duration of coronary perfusion was 15 minutes. Only the left coronary artery was perfused. The quality of heartbeat was much improved over that observed with retrograde perfusion, certainly an expected finding. Ventricular fibrillation did not occur in any experiment. At the outset we were concerned lest fibrillation be stimulated by whatever potential develops at the interface of oxygenated and nonoxygenated myocardium. The work by Brofman, Leighninger, and Beck[19] would indicate that ventricular fibrillation might be expected, particularly upon resumption of total cardiac perfusion. No serious arrythmias were noted.

Anatomic studies in man showed that forward perfusion with individual cannulation of both coronary ostia could be easily performed. Fig. 2 shows cannulation of both vessels, which would be advisable in human beings though nearly impossible, and perhaps unnecessary, in the dog. The ascending aorta in man fortunately is easier to work with than in dogs. One animal was lost from delayed hemorrhage at the aortic incision after a procedure remarkable for its difficulty in closing the aortotomy.

A point we believe to be of paramount importance is the use of a left atrial catheter to empty the left side of the heart. Visualization and cannulation of the coronary ostia is accomplished with great dispatch if a left atrial catheter is employed. Furthermore, at the conclusion of the procedure the left heart may be filled with blood or saline solution by means of the catheter to ensure displacement of all air. In dogs, we introduced a No. 18 Fr. plastic catheter into the left atrium through a pulmonary vein; the blood was returned to the oxygenator via the coronary drainage attachment. Decompression of the left side of the heart is essential when no septal defect exists. The operative field is nearly bloodless with removal of left atrial input.

## SUMMARY AND CONCLUSIONS

While the several forms of elective cardiac arrest may be applied to the surgical treatment of congenital aortic valvular disease under direct vision, some kind of coronary perfusion is necessary for the exposure of acquired diseases. Hearts with greatly decreased reserve are intolerant of any known type of arrest. Retrograde perfusion of the coronary sinus provides continuous myocardial oxygenation. Unfortunately, coronary sinus perfusion reaches principally only the left side of the heart, and the problem of cannula fixation remains incompletely solved. Distortions in the heartbeat induced by reversal of the coronary circulation also detract from the use of this technique.

Individual cannulation of the coronary ostia, after prompt emptying of the heart by means of the left atrial catheter, supplies an acceptable method for maintenance of cardiac circulation during direct approaches to the aortic valve. If anything, aortic insufficiency provides an easier field in which to work than does stenosis. A malleable metal, siliconized cannula with rubber cuff and multiple holes in the tip is suggested as an appropriate instrument for direct coronary ostial perfusion.

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# SCOLIOSIS AND HERNIATION AFTER OSTEOPLASTIC THORACOPLASTY

*Viking Olov Björk, M.D., and Roland Bunner, M.D.,*
*Uppsala and Stockholm, Sweden*

SINCE the introduction of osteoplastic thoracoplasty in 1952, it has been performed by us in 200 cases. The technique has been taken up in many chest centers, especially in Europe. Compared with other space-diminishing methods of the thoracic cavity as an addition to pulmonary resection for tuberculosis, it has the following advantages. (1) The size of the thoracic cavity can be tailored to fit the remaining lung parenchyma. (2) The fixation of ribs will leave a stable chest wall without paradoxical movements; thus, coughing is effective and ventilation unimpaired. The osteoplastic thoracoplasty can therefore be performed at the end of the pulmonary resection without increasing the risk for the patient. The advantage of a one-stage procedure is obvious. (3) The decrease in size of the thoracic cavity obtained at operation is permanent. (4) The functional result, both as regards pulmonary function and arm movements, is excellent. The cosmetic result is excellent and the deformity encountered is practically only visible in the axilla when the patient elevates his arms.

The aim of this paper is to emphasize important technical details and to report scoliosis and herniation of the lung in 104 patients from one to 5 years after the operation.

## HERNIATION OF THE LUNG ABOVE THE ROOF

The aim of the osteoplastic thoracoplasty is to obtain a permanent decrease of the thoracic cavity. Therefore, certain steps are taken to prevent a herniation of the lung above the roof.

1. The second rib is made as short as possible, thus, it will be straighter and it can be placed as close as possible to the mediastinum.

2. The costal cartilage is incised, facilitating fixation of the second rib to the mediastinum. This is a very important technical detail not performed in the beginning. When herniation was detected in some of the earlier cases

From the Thoracic Surgical Department, University Clinic, Uppsala, and the Roentgen Department, Sabbatsberg Hospital, Stockholm. Head: B. Holmgren, M.D.
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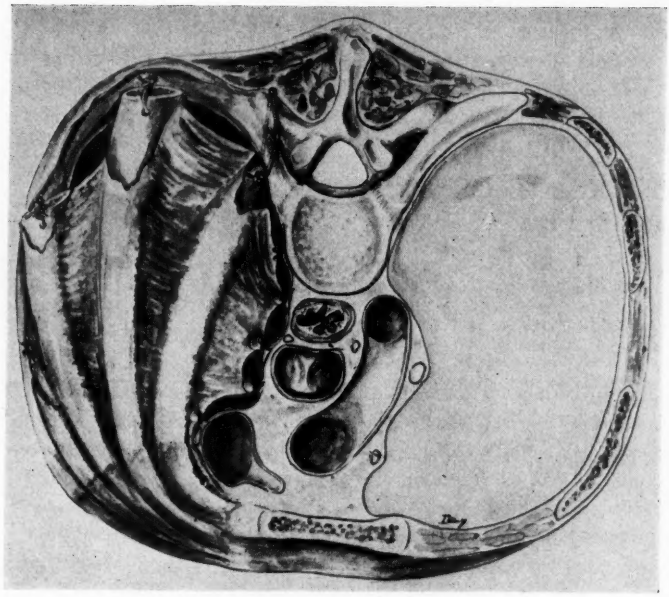


Fig. 1.—In the beginning, the second rib was bent in the costal cartilage and therefore a larger space resulted anteriorly between the rib and mediastinum causing herniation in many cases.

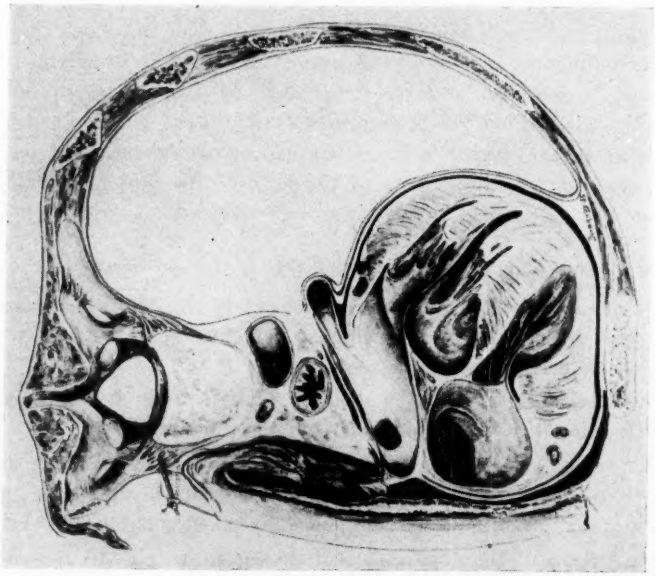


Fig. 2.—When the cartilage was incised anteriorly it was possible to place the second rib close to the mediastinum, preventing herniation.

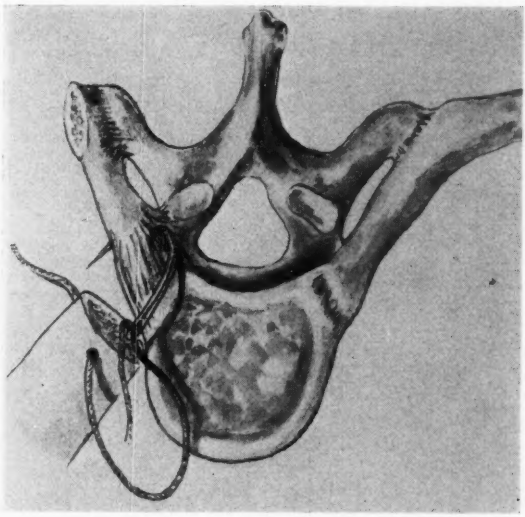


Fig. 3.—The posterior end of the rib is fixed as close to the midline as possible, i.e., by a suture through the costovertebral ligament.

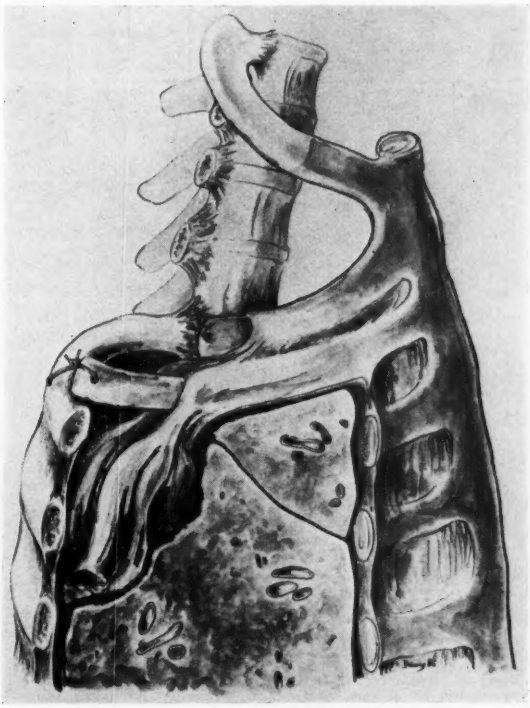


Fig. 4.—The second rib is posteriorly fixed to the vertebra by a suture through the costovertebral ligament.

and found to be anteriorly, this step was added and was found to decrease the incidence of herniation (Figs. 1 and 2).

3. The posterior end of the second rib is fixed by a nylon thread through a drill hole and then to the costovertebral ligament (Figs. 3 and 4).

4. The intercostal muscle bundle left on the top of the second rib is fixed to mediastinal tissue by a row of intercostal sutures, thus closing the roof of the chest medially (see Fig. 1).

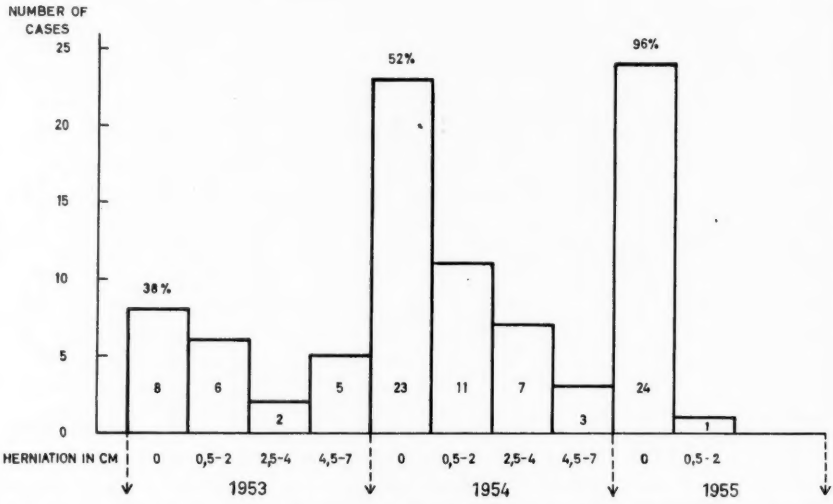



Fig. 5.—Diagram of the degree of herniation found in cases from the different years demonstrating the importance of technical details in preventing herniation.

Of the 104 cases investigated, 17 had the first rib removed; in all the other cases the first rib was left in place. There was the same incidence of herniation whether the first rib was left in place or not. Furthermore, the same incidence of herniation was found if the operation was carried out on the left or right side.

In the whole material the following degree of herniation was found:

| | |
|---|---|
| No herniation | 64% |
| The lung 0.5 to 2 cm. above the roof | 19% |
| The lung 2.5 to 4 cm. above the roof | 9% |
| The lung 4.5 to 7 cm. above the roof | 8% |

Fig. 6.—A roentgenogram of a 57-year-old man, who has had a right upper lobectomy and osteoplastic thoracoplasty. The second rib was not placed as close to mediastinum as possible, resulting in a big herniation. The hernia was 7 cm. above the roof. There was a tracheal deviation of 2 cm. and a scoliosis of 1 cm. over to the right side. The diaphragmatic movement was 5 cm. on the right and 7 cm. on the left side.

Fig. 7.—A roentgenogram of a 42-year-old woman, who had had resection of the superior lobe and superior segment of the lower lobe on the right side with an osteoplastic thoracoplasty. After 3 years this x-ray film was taken which demonstrates no herniation. a scoliosis of 2 cm., and deviation of the trachea of 4 cm. over to the right side. The right side of the diaphragm moved 6 cm., while the left side moved 7 cm. Brpnchospirometry showed an oxygen uptake of 23 per cent on the right side.

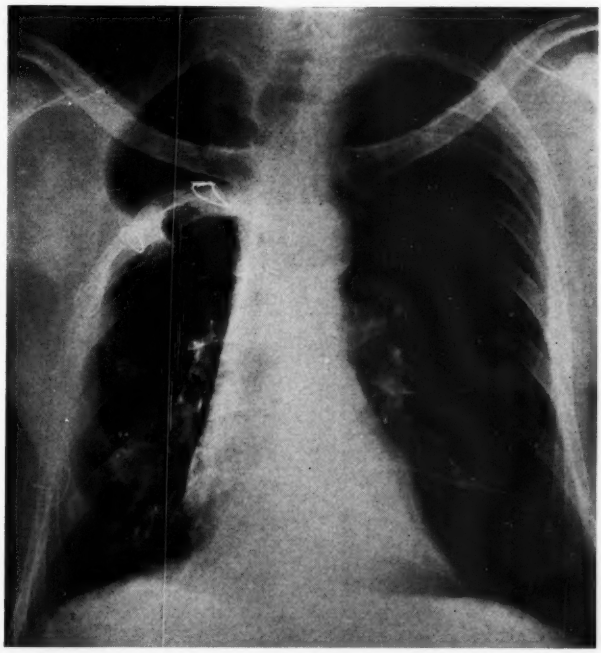


Fig. 6.

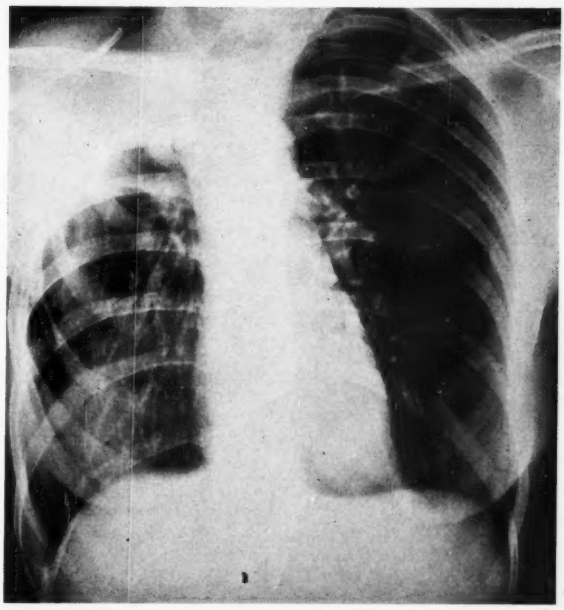


Fig. 7.

Figs. 6 and 7.—*For legends see opposite page.*

Fig. 8.

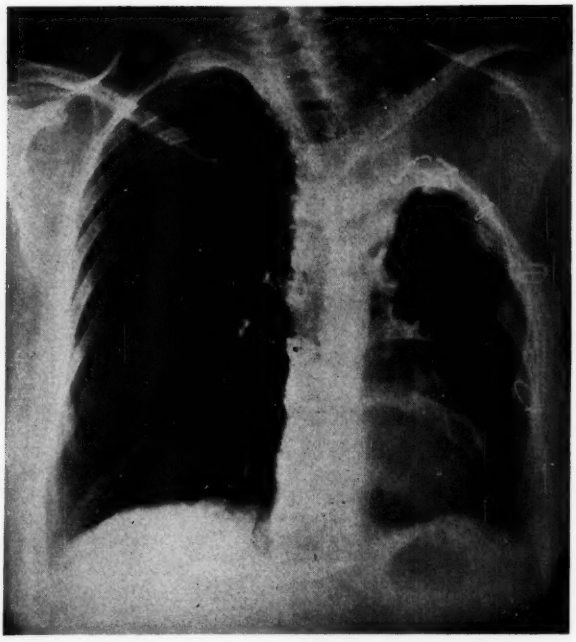
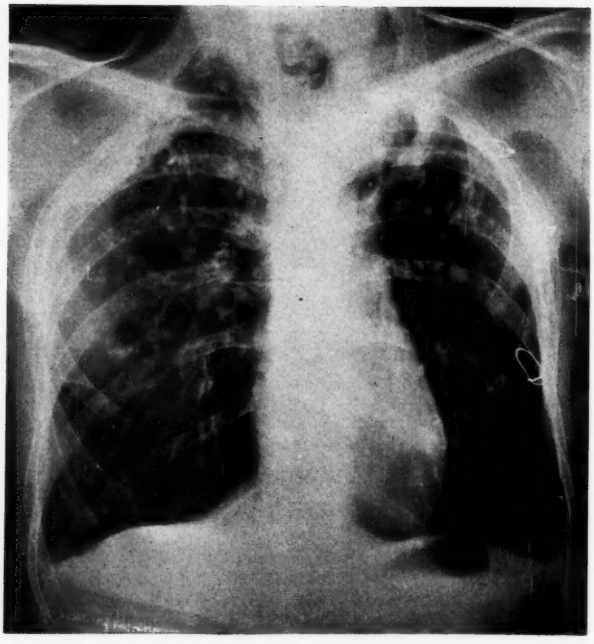


Fig. 9.



Figs. 8 and 9.—*For legends see opposite page.*

As some of the above-mentioned technical details were worked out during 1953, and the final technique adopted during 1954, it is interesting to see how important these technical details are in preventing herniation. Of patients operated on in 1953, only 38 per cent were without herniation, in

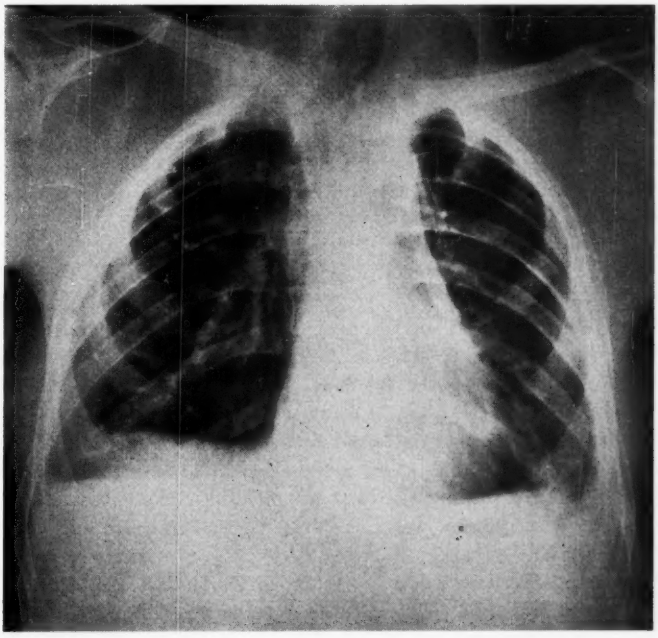


Fig. 10.—This x-ray film is of a 42-year-old man who has had bilateral resections with osteoplastic roof thoracoplasties on both sides. There is no herniation on either side. The first rib is left intact on both sides. There is a scoliosis of 1 cm. to the right side, a tracheal deviation of 1 cm. to the right side. The diaphragm moves 7 cm. on the right and 6 cm. on the left side. Three years after the operation the patient is in an excellent condition, working full time. Oxygen uptake on the right side, 57 per cent, on the left side, 43 per cent. Ventilation on the right side is 56, on the left, 44. Sputum and gastric washings are negative by guinea pig test.

the ones from 1954, 52 per cent, while in the cases from 1955 as many as 96 per cent were without herniation. The degree of herniation in cases from the different years will be shown in Figs. 5-10.

It can, therefore, be concluded that if the technique is followed in detail there will be a negligible incidence of herniation of lung above the roof.

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Fig. 8.—An x-ray film of a 39-year-old woman who had had resection of the superior lobe and the superior segment of the lower lobe on the left side plus osteoplastic thoracoplasty. After 3 years of observation there was no herniation above the roof, a scoliosis of 2 cm., and a tracheal deviation of 2 cm. over to the left side. The left side of the diaphragm moved 6 cm. while the right side moved 8 cm.

Fig. 9.—A roentgenogram of a 41-year-old man, who had had a right conventional thoracoplasty with apicolysis 5 years previously. The film shows a re-expansion of the right upper lobe above the clavicle. On the left side, on the other hand, the film shows the results of a resection of the apicoposterior and anterior segments of the left upper lobe plus the superior segment of the lower lobe with an osteoplastic thoracoplasty performed 4 years previously. Four years after this operation, there is no herniation above the roof, there is no scoliosis but a tracheal deviation of 1.5 cm. over to the left. The left and right sides of the diaphragm move equally, 5 cm.

SCOLIOSIS

There was no difference in the degree of scoliosis whether the first rib was left in place or resected. To measure the amount of scoliosis, a line was drawn on the frontal roentgenogram between the midpoint of the first and twelfth thoracic vertebra.

The maximal deviation from this line was measured and the following values obtained:

| | |
|---|---|
| No scoliosis | 13% |
| 0.5 to 1 cm. deviation | 43% |
| 1.5 to 2 cm. deviation | 36% |
| 2.5 to 3 cm. deviation | 8% |

Some scoliosis was therefore found in 87 per cent of the cases. In most of these cases there was only a slight scoliosis, 43 per cent. A moderate scoliosis was encountered in 36 per cent. A severe scoliosis was only found in 8 per cent.

*Tracheal Deviation.*—The tracheal deviation was measured in centimeters from the same line between the first and the twelfth thoracic vertebra used for calculation of the scoliosis. Usually there was a tracheal deviation to the operated side.

The tracheal deviation was found to be somewhat smaller if the first rib was resected on the right side, on an average of 1.8 cm. as compared with 2 cm. for cases where the first rib was left behind. The corresponding values on the left side were 1.1 cm. when the first rib was resected and 1.7 cm. when it was left behind.

Furthermore, there was less tracheal deviation if the patient was operated upon on the left side. This is to be expected as the position of the aorta will diminish the deviation of the trachea to the left side. The deviation of the trachea was always to the side of operation with the exception of one case in which a contralateral fibrothorax kept the trachea fixed 2 cm. to the opposite side of the operation.

There was some correlation between the tracheal deviation and the scoliosis. A more pronounced scoliosis was usually accompanied by a greater deviation of the trachea (Table I).

TABLE I. THE CORRELATION BETWEEN THE SCOLIOSIS AND THE DEVIATION OF TRACHEA AFTER AN OSTEOPLASTIC THORACOPLASTY

| NO. OF CASES | SCOLIOSIS (CM.) | TRACHEAL DEVIATION (CM.) | |
|---|---|---|---|
| | | AVERAGE | RANGE |
| 13 | 0 | 1.3 | 0-2.5 |
| 39 | 0.5-1.0 | 1.4 | 0-3 |
| 39 | 1.5-2.0 | 2.0 | 0-4 |
| 8 | 2.5-3.0 | 2.3 | 1-3 |

*Diaphragmatic Movements.*—No severe impairment of the diaphragmatic movements was encountered after pulmonary resection with an osteoplastic thoracoplasty. After right-sided operation, the maximal diaphragmatic movements were on the right side on an average 4.5 cm., range from 2 to 9.5 cm.,

as compared with an average of 5.6 cm., range from 2 to 9 cm. After a left-sided operation, the left diaphragm moved on an average of 4.6 cm., range from 2 to 7 cm., as compared with the right which on an average moved 5.8 cm., range from 3 to 10 cm.

SUMMARY

A follow-up study has been carried out on 104 patients undergoing pulmonary resection with an osteoplastic thoracoplasty in order to estimate the degree of scoliosis, herniation, tracheal deviation, and the diaphragmatic movements. In the beginning, a herniation of the lung above the roof was encountered rather frequently. When the technique was further developed, with incision in the second costal cartilage and a fixation of the second intercostal muscle bundle to the mediastinal pleura, the incidence of herniation became negligible.

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# FUNCTIONAL STENOSIS DUE TO BRONCHIAL ANGULATION

*C. H. Nicholson, M.D., J. B. Perkins, M.D., E. B. Emerson, Jr., M.D., and G. L. Emerson, M.D., Batavia, N. Y.*

A SIGNIFICANT consideration in determining the extent of pulmonary resection for tuberculosis is the presence or absence of bronchial stenosis. Functional stenosis due to kinking of the bronchus by extrinsic disease may resemble endoscopically the fixed stenosis due to intrinsic thickening.

Björk in a series of 1,000 resections reported 4 cases with a diagnosis of apparent bronchial stenosis. They were associated with lower lung field disease causing a "dislocation of the lung by pleural effusion and fibrosis." At exploration, one with bronchotomy, the apparent stenosis was found to be due to simple kinking of the bronchus.

In a series of 200 resection cases, we noted 4 patients with destroyed left upper lobes who showed bronchial angulation which simulated anatomic stenosis at the time of preoperative bronchoscopy. On first inspection of the bronchus, it appeared that intrinsic stenosis was present and that pneumonectomy would be indicated. Such a course was to be avoided if possible as these patients generally have contralateral disease and a limited pulmonary reserve. To preserve the large volume of parenchyma in the lower lobe, it was important to recognize that the deformity was due mainly to extrinsic factors.

Bronchograms of 3 of these, and of another who refused surgery, illustrated the typical deformity pictorially (Fig. 1). The characteristic finding was a left main bronchus which curved outward and then upward, leading directly into the upper lobe orifice rather than the lower. The lower lobe took off from this upward curve in a sharply posterior and downward direction, producing marked angulation. Tuberculous bronchiectasis was present in the upper lobe in each case, and in the superior segment in one. No significant basilar bronchiectasis due to poor drainage was evident. Serial tomograms showed that the bronchial deformity was not present during the acute stage of the disease but developed as the destroyed left upper lobe contracted.

The sputum was persistently positive and resistant to isonicotinic acid hydrazide and streptomycin in 3 cases at the time of surgery. The pulmonary findings by x-ray and exploration in each case were those of a contracted,

From the Surgical Service, Veterans Administration Hospital, Batavia, N. Y., and the University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

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destroyed left upper lobe with open cavitation, an overdistended lower lobe, and pleural and peribronchial fixation by fibrosis. A left upper lobectomy, with combined superior segmentectomy in one, and an attempt to free the angulated bronchus was carried out in each case. The postoperative course

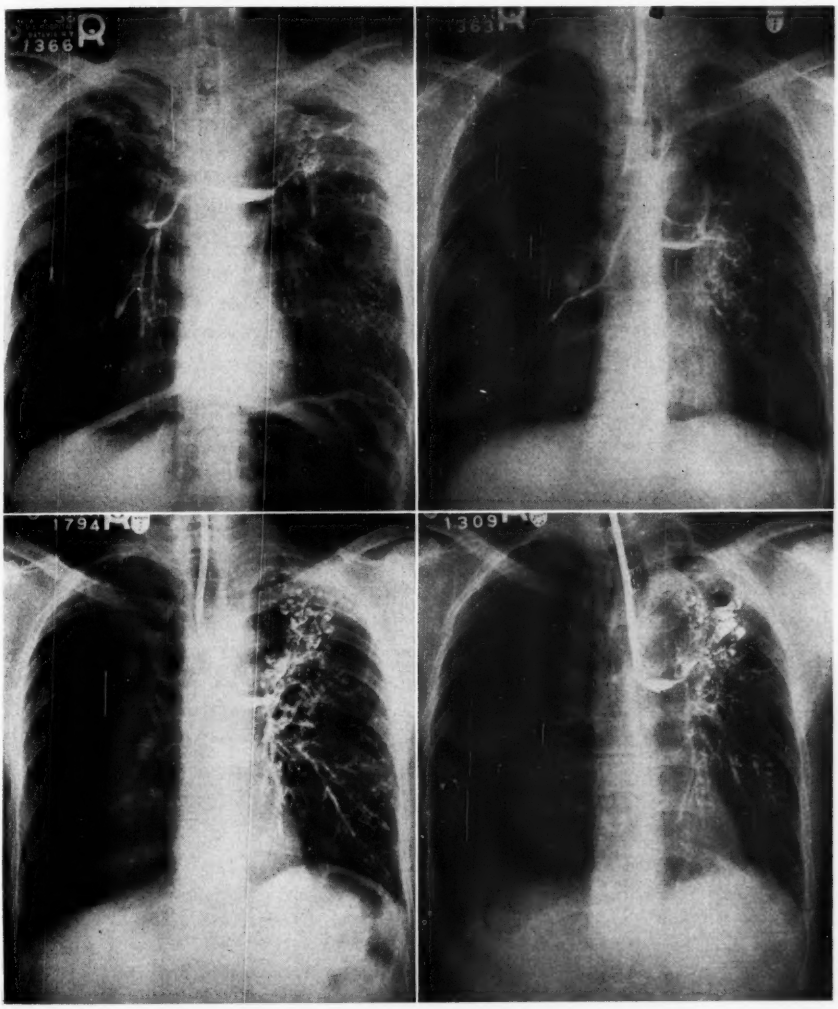


Fig. 1.—Bronchograms of 4 patients which illustrate upper lobe cavitation and bronchiectasis associated with angulation of the lower lobe take-off.

was complicated in 2 cases by bronchopleural fistula, in one by staphylococcal empyema, and in one by atelectasis. Postresection thoracoplasty was added in each. These patients recovered slowly but completely.

During a 2- to 4-year follow-up, x-ray studies have revealed a stable lung and no positive cultures have been obtained. Results are considered good for control of the tuberculosis. Recent bronchograms of 3 patients show partial correction of the functional stenosis and no evidence of basilar bronchiectasis. One patient has developed cylindrical stenosis of the lower lobe bronchus. Clinically, they have all been quite free of cough or sputum production.

SUMMARY

A destroyed, contracted left upper lobe may be associated with sufficient angulation of the lower lobe bronchus to cause functional stenosis. This is easily confused endoscopically with the anatomic stenosis produced by intrinsic bronchial disease. Recognition of this condition may allow conservation of pulmonary tissue at operation. Four such cases are reported.

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# RUPTURES IN THE BRONCHIAL TREE DUE TO BLUNT TRAUMA

*Elmore M. Aronstam, Lieutenant Colonel, MC, USA, James G. Inman, Colonel, MC, USA, and Franklin Mitchell, Captain, MC, USA, Neubrucke, Germany*

MUCH progress in the handling of chest injuries has occurred since the beginning of World War II. Similarly, many improved surgical and anesthetic techniques have been developed to help cope with the more serious injuries, such as those to the trachea and bronchi.

Blunt injuries to the thorax have been implicated as the cause of many pathologic states: diaphragmatic lacerations, intrapulmonary hematomas, and cavities, lacerations of the great vessels, and ruptures of the trachea or bronchi. The latter type of injury has been rather rare. Nevertheless, it is becoming apparent that with early diagnosis and proper management, patients with these injuries can be restored to full activity.

A review by Kinsella and Johnsrud in 1947[1] demonstrated a 33 per cent mortality associated with these conditions. Secondary repair in patients who luckily survive the injury is extremely difficult and plagued by the problems of bronchostenosis and grafting. It is obvious that ideal treatment would be prompt surgical repair. We have found only three reports of successful primary operations. Griffith[2] reported such an operation in 1949, Scannell[3] primarily repaired a laceration of the thoracic trachea, reporting it in 1951. Richards and Cohn[4] reported a similar case in August, 1955. These patients are indeed desperately ill but when anesthesia is skillfully handled, corrective surgery can be accomplished. Recent experiences with 2 desperately injured patients justify this point of view.

## CASE REPORTS

CASE 1.—A 25-year-old airman was struck by the steering post of his automobile in a head-on collision. He received emergency treatment at a nearby hospital, where he was found to have severe dyspnea with extensive subcutaneous emphysema over neck, chest, abdomen, and arms. A right tension pneumothorax was present; a small catheter was inserted into the right chest and connected to the underwater-seal bottle (Fig. 1). As leakage of air was tremendous and continuous even during expiration, a bronchial tear was suspected. The patient was quickly transported to a general hospital, where bronchoscopy was performed under topical anesthesia. The bronchoscope passed into the mediastinum through a rent at the bifurcation of the trachea. As visualization was

From the Thoracic and Anesthesia Services, 98th General Hospital, Neubrucke, Germany. Received for publication Dec. 5, 1958.

impaired, the scope was quickly withdrawn. The patient was adequately ventilated with oxygen and was then rapidly intubated, utilizing minimal amount of Pentothal followed by Anectine. A Carlen's endobronchial catheter was utilized, enabling the anesthesiologist to ventilate either lung and proved to be of great value in the almost constant bronchial aspiration that was necessary to remove the secretions. A standard right thoracotomy

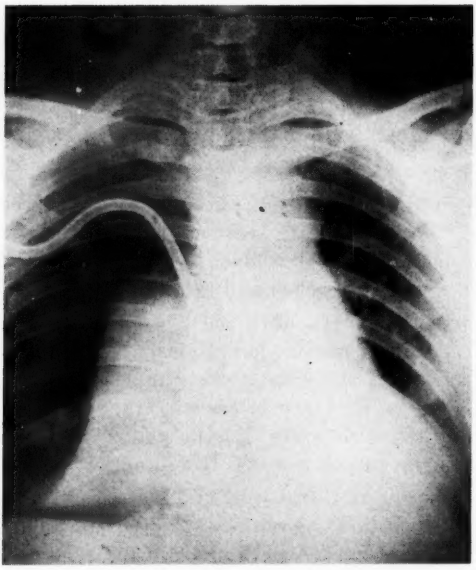

Fig. 1.—Case 1. Preoperative x-ray film.

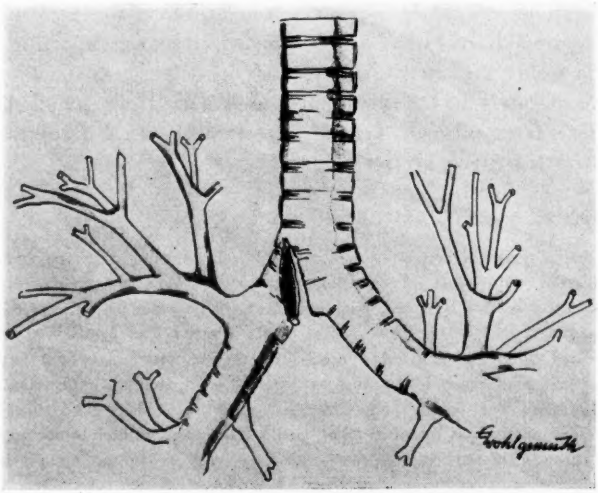

Fig. 2.—Case 1. Tracheal tear.

was performed. On entering the chest, a tension pneumothorax (present despite the catheter drainage) was relieved. Air escaped violently into the pleural cavity from an opening into the mediastinum between the inferior and superior pulmonary veins. This was tamponaded with a lap pad to permit partial inflation of the right lung. The posterior mediastinum was opened and the azygos vein divided, exposing the trachea. The circumferential tear through the carina had almost completely separated the right main stem from the trachea (Fig. 2). The Carlen's catheter was inserted into the left main bronchus, its balloon inflated, and the trachea repaired with interrupted fine silk sutures.

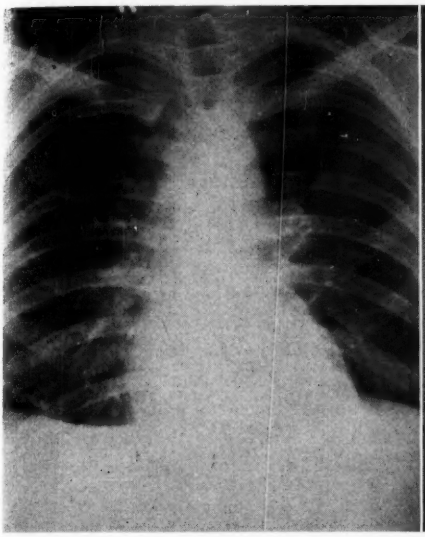

Fig. 3.

Fig. 4.

Fig. 3.—Case 1. Postoperative x-ray film.
Fig. 4.—Case 1. Late postoperative x-ray film.

It was noted that the right lung felt extremely crepitant. This was a coarse crepitus, similar to that felt in the skin. A fair degree of interstitial emphysema was presumed to be present. The anesthesiologist commented upon the resistance of the lungs to his manual ventilation despite a clear airway. The chest was closed with underwater-seal catheter drainage and a tracheotomy was performed (Fig. 3). The patient gradually became less and less short of breath during the week following operation. He was bronchoscoped one month following surgery (Fig. 4). The trachea was then well healed with no evidence of stenosis. The patient was returned to full duty.

CASE 2.—This patient sustained a similar blunt injury in an automobile accident. He was hospitalized promptly and found to be severely dyspneic with emphysema of the neck, shoulders, left side of the chest and abdomen. A tracheotomy was performed. However, dyspnea did not abate and emphysema became more widespread. A left pneumothorax was noted. A catheter was placed anteriorly in the left chest and a tension pneumothorax partly relieved. The right lung remained fully expanded throughout the course. Consultation was then obtained as the patient did not improve. Twenty-four hours after injury the patient was operated upon. After a small dosage of Pentothal and Anectine, a Carlen's catheter was rapidly inserted into the trachea and the left chest opened through a standard incision. A tension pneumothorax was present. The patient

had sustained a deep laceration of the lingula, tearing a segmental branch from which a sizable air leak occurred. A small intrapulmonary hematoma was present in the lower lung. In addition, the lung was extremely crepitant, again indicating interstitial emphysema. The anesthesiologist again commented on the difficulty of ventilating this patient. The torn bronchus was ligated and covered with adjacent pulmonary parenchyma. Throughout this manipulation, the anesthesiologist experienced difficulty in clearing the left-main stem bronchus of dark blood. As exploration of the mediastinum (carina and main-stem bronchus) showed no further damage, the chest was closed after insertion of catheters. The tracheotomy wound was opened and a small thyroid ima artery was found to be open and bleeding into the trachea. This was ligated and a tracheotomy tube was re-inserted as the Carlen's catheter was withdrawn. As expected, the patient was improved, but still dyspneic for several days following surgery. However, he eventually made a very satisfactory recovery from his injury.

## SUMMARY

It is our firm belief that prompt clinical recognition of bronchial tears permitted successful surgery in these patients. The presence of subcutaneous emphysema, dyspnea, and tension pneumothorax were the presenting symptoms and the key to diagnosis. Early operative efforts were rewarded by good healing without serious sequelae (stenosis, atelectasis, and infection). The Carlen's catheter was most useful in permitting ventilation of one lung while the other was out of the surgical field. The successful anesthetic management in each case, rapid intubation under Pentothal and Anectine anesthesia lent itself well to the problems at hand. The presence of interstitial emphysema in the lungs was noted with interest. Certainly it delayed return of adequate ventilation for several days following surgery and tracheotomy was most helpful in this critical situation.

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# A NEW TECHNIQUE FOR THORACOTOMY AND CHEST CLOSURE IN THE EXPERIMENTAL ANIMAL

*Robert A. Salerno, M.D., New York, N. Y.*

RECENT advances in the surgical treatment of cardiovascular diseases have been attended by a marked increase in heart and blood vessel research using the dog as the experimental subject. Observation in many laboratories suggests the need for more careful technique in the thoracotomy and chest closure and for more stringent application of basic surgical principles to the experimental animal. More specifically, chest incisions in the dog frequently seem ill placed and too large, and often involve unnecessary structures. Because of the thoracotomy which is less than ideal, the closure is consequently more difficult, less secure, and more susceptible to postoperative complications.

In addition to the primary requisite of careful surgical technique, what seems needed is an understanding of the comparative anatomy of the thorax of man and dog, so that incisions can be adapted to the anatomic structures as carefully in the animal as they are in man. The object of this paper is to review certain pertinent musculoskeletal aspects of canine chest anatomy and to describe a technique for thoracotomy and closure which gives ample exposure for most cardiac operations, and has proved to be quick, easy, and effective.

## COMPARATIVE ANATOMY

The thoracic anatomy peculiar to the dog is best appreciated in the light of his physiologic requirements. The dog has problems of weight distribution, locomotion, and thoracic respiration which do not exist in the primate. As might be expected, these different physiologic requirements are reflected in several basic anatomic differences between dog and man. Some of the more important of these differences can be summarized as follows.[1, 4]

*Skeletal.—*

1. *Vertebrae and ribs:* The dog has 13 thoracic vertebrae and corresponding ribs, the upper 9 of which articulate directly with the sternum, the next 3 uniting to form a cartilaginous costal arch, and the thirteenth rib "floating" without anterior articulation. This is in contrast with the 12 vertebrocostal units of man, 7 pairs of which articulate with the sternum, 3 forming a

From the Department of Surgery, Columbia University, College of Physicians and Surgeons, and Presbyterian Hospital, New York, N. Y.

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chondral union, and 2 "floating" anteriorly. Despite the fact that the dog has a greater number of ribs attaching to the sternum, his costochondral attachments and therefore his entire rib cage are more flexible than man's. This greater flexibility is an anatomic adaptation to the dog's need for a proportionately greater vital capacity to accommodate running. It is this same greater costochondral flexibility that allows wide intercostal exposure in the thoracotomy without the need for rib resection.

2. *Clavicle:* In the dog there is no clavicle interposed between the acromion of the scapula and the manubrium sterni. In forming the anterior portion of the shoulder girdle in man, the clavicle has nearly a dozen muscular and ligamentous attachments. With the absence of the clavicle in the dog, the anatomic relationships of these attachments are distinctly altered.

*Muscular.—*

1. *Scalenus:* In both dog and man, the scalenus is primarily an accessory muscle of inspiration, arising from the transverse processes of the lower 4 to 6 cervical vertebrae. As in the human being, the scalenus of the dog forms three distinct muscle bundles, *anticus, medius,* and *posticus.* In man, the scalenus *anticus* and *medius* insert on the inner and upper surfaces of the first rib, respectively, while the *posticus* inserts on the outer surface of the second rib. In the canine, the *anticus* (*scalenus primae costae*) similarly inserts onto the first rib. The *posticus* of the dog, however, has its inferior attachment on the fourth rib. Most important for this discussion is the *medius,* the largest and longest portion of the scalenus, which inserts onto ribs 5 through 9 via an extensive aponeurosis partly hidden by the overlying *external abdominal oblique.*

2. *Serratus anterior (ventralis):* In man, this muscle is a thin muscular sheet arising by fleshy digitations from the outer surface and superior borders of the upper 8 or 9 ribs, and inserting onto the vertebral aspect of the scapula. Its action in man is primarily rotation of the scapula as occurs with raising and lowering of the shoulder and with pushing. In the dog, the serratus anterior is much more highly developed and more powerful, since the right and left serratus muscles form a muscular "sling," supporting the weight of the trunk onto the pectoral limbs, especially during running or leaping. For this greater function the muscle has a greater trunk distribution, arising from the transverse processes of the lower 5 cervical vertebrae as well as from the lateral surfaces of the upper 8 or 9 ribs.

3. *Rectus abdominus:* In both man and dog, this muscle originates at the pubic crests. In man, the rectus extends superiorly to insert on the anterior cartilaginous portions of ribs 5, 6, and 7. In the dog, the rectus traverses the entire thoracic cage to insert superiorly onto the first rib. This longer distribution in the dog allows the rectus to aid in the advancement of the hind limbs during gallop.

Some of the more important relationships of these muscles are depicted in Fig. 1. Occasionally variations from the normal will occur. In two experiments, for example, the scalenus medius had its insertion begin on rib 6 rather

than rib 5. Since this muscle is a particularly important landmark, its exact distribution should be ascertained in each instance. It should be noted, furthermore, that the *latissimus dorsi*, the *external abdominal oblique*, and the *intercostal muscles* of the dog are essentially similar in origin, distribution, and function to the corresponding muscles in man.

### THE THORACOTOMY

Anesthesia is induced by intravenous sodium Pentothal, and an endotracheal tube is inserted and connected to a suitable respirator. The dog is placed in the completely lateral position, with a pack of towels, 2 or 3 inches in thickness, inserted under the table side of the animal. This arches the operative side into better view, and allows the shoulder girdle to fall forward out of the field of incision.

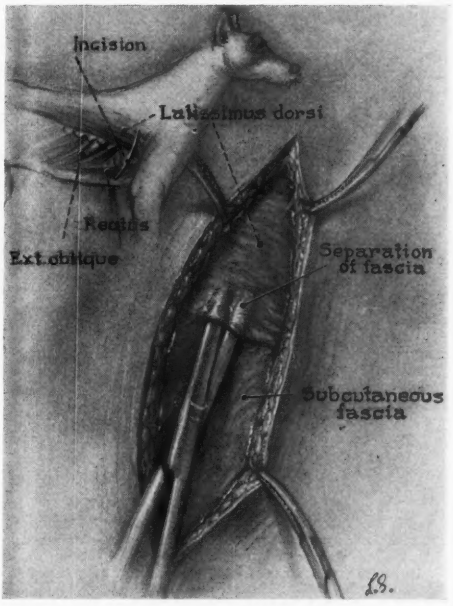


Fig. 1.—The skin and subcutaneous tissue are incised. A small incision is made in the subcutaneous fascia at its function with the latissimus dorsi.

The ribs, the latissimus dorsi, and rectus abdominus are palpated and the area of the fourth intercostal space is identified. Incision is made through the skin and subcutaneous tissue over the fourth intercostal space, some 12 cm. in length, and extending from approximately 4 cm. dorsal to the lateral border of the latissimus dorsi ventrally to the lateral border of the rectus abdominus. At this juncture the lateral border of the latissimus dorsi is identified occupying the upper one third to one half of the wound bed (Fig. 1) with the subcutaneous fascia extending ventrally from its lateral margin.

A small incision, 1 cm. in length, is made in the subcutaneous fascia at its junction with the latissimus dorsi. Both index fingers are inserted into this opening and by gentle traction in opposite directions (cephalad and caudad) the fascia is separated from its muscular attachment.

Three important landmarks are now seen (Fig. 2, *A*): (1) the long and narrow scalenus medius, (2) the large digitations of the serratus anterior above the scalenus, and (3) the thin superior margin of the external abdominal oblique overlying the beginning of the scalenus medius insertion onto the fifth rib.

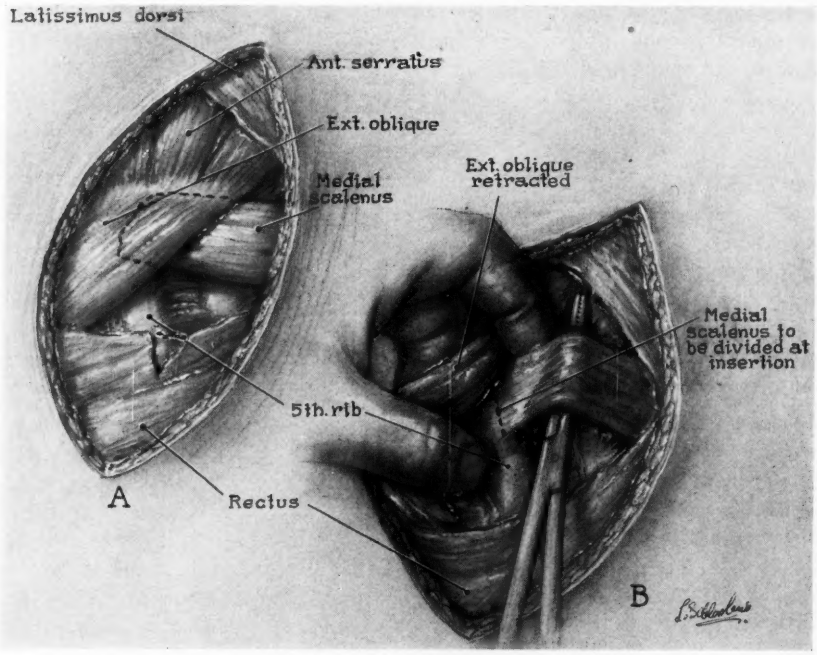


Fig. 2.—*A*, Three important landmarks are brought into view: the scalenus medius, the serratus anterior, and the external abdominal oblique.

*B*, The aponeurosis of the medial scalenus is put on stretch with a straight clamp prior to division of its aponeurosis at its insertion onto the fifth rib.

A straight clamp is inserted under the scalenus medius and is retracted caudad, lifting the aponeurosis from its rib attachment. The stretched avascular aponeurosis (Fig. 2, *B*) is transected, allowing the scalenus medius to fall cephalad with ease, and exposing the fourth intercostal space. It should be noted that exposure to the intercostal muscles is achieved completely by muscle retraction. Neither the latissimus dorsi, the external oblique, the serratus anterior, nor the scalenus medius need be transected. In order to avoid bloody transection of the intercostals, the full jaw length of a straight

clamp is first inserted under the fibers of the external intercostal muscle midway in the intercostal space. The muscle fibers are than separated by either incision between the clamp jaws, or by gentle traction of the clamp toward the skin (Fig. 3, *A*). The internal intercostal muscle is similarly divided.

The parietal pleura is now visualized. During the expiratory phase of respiration, the pleura is opened. The fourth and fifth ribs are then separated by the gradual opening of a self-retaining retractor. At this point, the digitations of the serratus anterior are seen as thick muscle bundles straddling the intercostal space in the upper portion of the wound; the rectus abdominus is identified traversing the lower aspect of the wound. If necessary these muscles may be split in the direction of their fibers to obtain greater exposure.

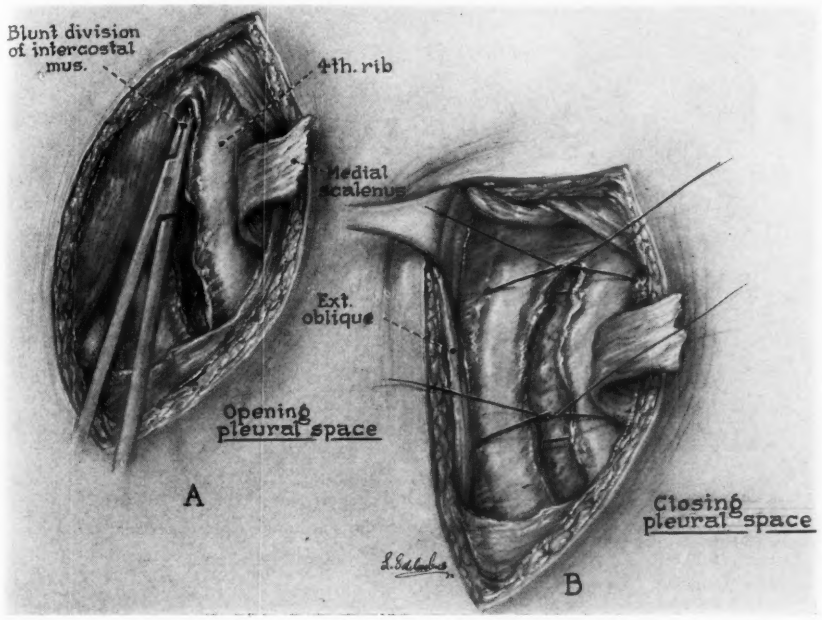


Fig. 3.—*A*, The intercostal muscles are divided midway in the fourth interspace. *B*, Closure of the thoracotomy is begun by approximating the fourth and fifth ribs with two pericostal silk sutures.

THE CLOSURE

The thoracotomy closure is effected in five layers. (1) Two pericostal sutures of heavy silk are used to reapproximate the fourth and fifth ribs (Fig. 3, *B*). (2) A silk suture is placed dorsally and deeply into the thick bundles of the serratus anterior. This suture is carried in continuous fashion ventrally to about the midpoint of the wound. The raised flap of the scalenus medius is then sutured to the superior margin and anterior surface of the external abdominal oblique (Fig. 4, *A*). (3) The edge of the latissimus dorsi

muscle is now reapproximated to the subcutaneous fascia (Fig. 4, *B*), preferably with interrupted silk sutures. (4) The subcutaneous tissue, and (5) the skin are closed in layers. A rubber catheter can be placed in the pleural space and brought out through the wound in order to allow aspiration of air. The catheter is removed before the final suture is set in place.

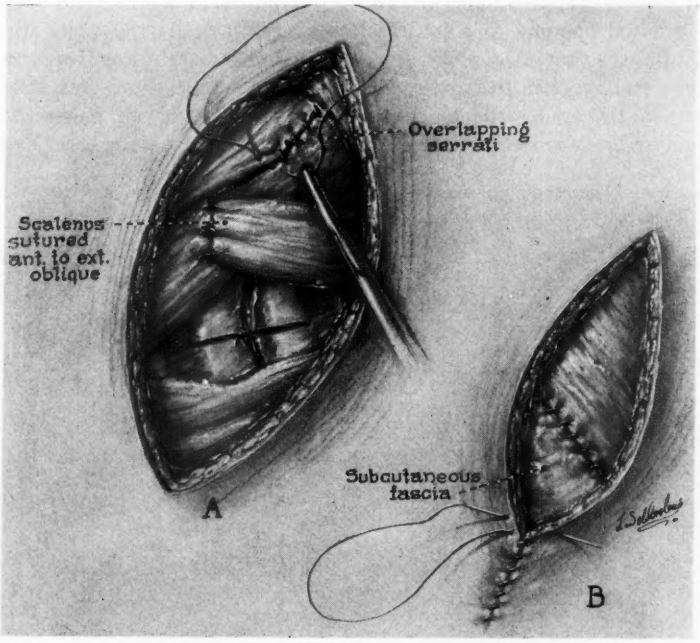


Fig. 4.—*A*, The thick bundles of the serratus anterior overriding the fourth interspace are reapproximated. The scalenus medius is sutured to the antero-superior margin of the external abdominal oblique.

*B*, The latissimus dorsi is reapproximated to the subcutaneous fascia. The subcutaneous tissue and skin are closed.

## DISCUSSION

In other laboratories, the latissimus and longissimus dorsi, the scalenus, the serratus anterior, the external abdominal oblique, and the rectus abdominus are variously incised. Many operators routinely employ subperiosteal rib resection.[2, 3] It is our experience that in the large majority of experimental chest procedures such extensive surgery is unnecessary; it adds to the operating time; it entails significant blood loss and trauma; it involves the use of large amounts of foreign body silk; and it makes the closure less secure.

In over 150 dog operations, this new technique has been used without a single wound complication. The operating time usually does not exceed 3 minutes for the thoracotomy and 6 minutes for the closure. There is usually no bleeding except in the incision of the skin and subcutaneous fat, and little or no silk is needed for hemostasis below this level.

SUMMARY

Major emphasis is placed upon careful technique and a thorough understanding of the anatomy as prerequisites to good thoracic surgery in the experimental animal. In the canine, the absence of the clavicle alters the structural relationships of a number of muscular and ligamentous attachments, particularly that of the scalenus medius which becomes an important landmark in the thoracotomy.

With this anatomy in mind, a new technique for thoracotomy is developed in which retraction of muscle and fascia is the predominant feature. The latissimus dorsi and the scalenus medius are separated from their local fascial and aponeurotic attachments. These muscles and the adjacent serratus anterior and rectus abdominus are then retracted to allow wide exposure to the intercostal space without rib resection. Thus, transection of all major muscles is completely avoided. Because of the careful thoracotomy, the closure is made a simple and secure procedure of reapproximation.

This new approach has been employed in over 150 cardiac experiments without a single wound complication. The method has proved to be quick and easy, avoiding unnecessary blood loss and trauma, and ultimately facilitating the post operative recovery.

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# A STUDY OF DIAPHRAGMATIC AND CRURAL INNERVATION BY THE DIRECT RECORDING OF ACTION POTENTIALS IN THE DOG

*Anthony DiBenedetto, M.D., Arthur A. Siebens, M.D., John J. Cincotti, M.D., Austin R. Grant, M.D., and Paul Glass, B.S., Brooklyn, N. Y.*

## INTRODUCTION

SURGERY for hiatus hernia and its related problems has attracted interest in the innervation of the diaphragm and its component parts surrounding the esophagus. Collis and his associates[1] have suggested that in performing reparative surgery of hiatal hernias, the nerve supply to the left crus may be interrupted. His interest in this problem stimulated him and his co-workers to make a thorough anatomic study of the nerve supply to this region of the body.[2] According to this work, the right crus was invariably innervated by the right phrenic nerve and the left crus by the left phrenic nerve. Botha[3] extended these studies to include a variety of animals, including the dog, and found no evidence of contralateral crural innervation either by anatomic technique or following phrenic nerve stimulation. Ogawa and co-workers,[4] however, recently described crossed innervation of the dogs' right crus in approximately 30 per cent of the animals studied.

Because the difficulties of visually tracing fine nerve twigs from origin to termination are considerable, a physiologic approach to the problem of crural innervation was thought to have certain advantages. In the present study, the criterion of innervation was an action potential picked up from crura or diaphragms when either phrenic nerve was stimulated by single electrical pulses.

## METHOD

Eleven mongrel dogs were anesthetized with Nembutal and intubated. A Pneophore* demand-type respirator was attached to the endotracheal tube to provide ventilation after the thorax was opened. A midline incision was made from just below the umbilicus to the sternal notch and the sternum was split

From the Cardiopulmonary Laboratory and the Thoracic Surgical Section of the Brooklyn Veterans Administration Hospital, Brooklyn, N. Y., and the Department of Physiology, State University of New York, Downstate Medical Center, Brooklyn, N. Y.

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*The Mines Safety Appliance Co., Pittsburgh, Pa.

in the midline. Both phrenic nerves were dissected from the mediastinum. The liver was then removed as described by LeVeen and Lewis[5] and a plastic (Tygon) cannula with an attached side arm was interposed between the superior and inferior caval veins. The portal vein was then attached to the side arm. This system allowed restoration of caval flow from the inferior vena cava and portal system (Fig. 1). The animal was immediately heparinized. With the removal of the liver, the crura could be clearly visualized. Plastic electrodes with imbedded silver wire contact points were then accurately attached by fine suture to each crus and each diaphragmatic leaf (Fig. 1). Contact points of

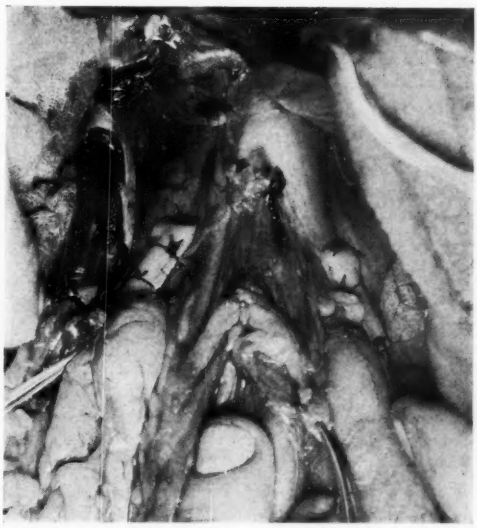

Fig. 1.—Photograph of crural electrodes in position. The animal's chest is located superiorly, the abdomen inferiorly. The cut edge of the diaphragm extends diagonally across the upper part of the field. The liver has been removed. Each electrode is on crural muscle. The esophagus lies between the crura. To the right of the dog's right crus is the cannula retaining continuity of the inferior vena cava and portal vein.

these recording electrodes were 0.5 mm. in diameter and 3 mm. apart. The previously mobilized phrenic nerves were sectioned 4 inches from their termination and the distal ends passed through stimulating electrodes. A variable frequency stimulator was used for stimulating nerves and a 4-channel direct recorder for recording action potentials. With this arrangement, action potentials could be recorded simultaneously from both diaphragmatic leaves and both crura. The phrenic nerves were stimulated by single pulses.

## RESULTS

In each of the 11 animals, action potentials were recorded from the right diaphragm and right crus when the right phrenic nerve was stimulated, and from the left diaphragm and left crus when the left phrenic nerve was stimulated (Table I, Fig. 2). In 4 animals, action potentials were also recorded from

the right crus when the left or contralateral phrenic nerve was stimulated. There was no instance in which action potentials from the left crus followed stimulation of the right phrenic nerve. Thus, although the left crus was only innervated by the ipsilateral nerve, it was not unusual for the right crus to be innervated by both phrenic nerves.

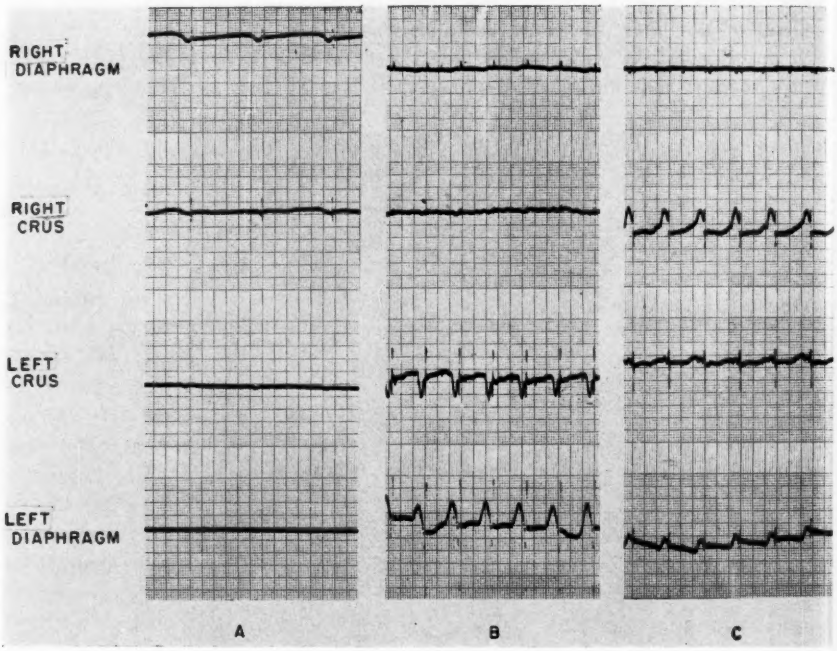


Fig. 2.—Action potentials recorded from crura and diaphragm when right (*A*) and left (*B* and *C*) phrenic nerves were stimulated by single pulses. Records *A* and *B* illustrate ipsilateral innervation only. Record *C* shows crossed innervation of the right crus by the left phrenic nerve.

TABLE I

| EXPERIMENT | STIMULATION OF RIGHT PHRENIC NERVE ACTION POTENTIALS FROM | | | | STIMULATION OF LEFT PHRENIC NERVE ACTION POTENTIALS FROM | | | |
|---|---|---|---|---|---|---|---|---|
| | RD | RC | LD | LC | RD | RC | LD | LC |
| 3/29/57 | + | + | +* | – | – | + | + | + |
| 5/ 8/57 | + | + | – | – | – | + | + | + |
| 5/20/57 | + | + | – | – | – | – | + | + |
| 6/24/57 | + | + | – | – | – | – | + | + |
| 7/12/57 | + | + | +* | – | +* | – | + | + |
| 9/13/57 | + | + | +* | – | – | + | + | + |
| 9/27/57 | + | + | – | – | – | – | + | + |
| 11/ 1/57 | + | + | – | – | – | – | + | + |
| 11/ 8/57 | + | + | – | – | +* | – | + | + |
| 11/15/57 | + | + | – | – | – | + | + | + |
| 12/ 5/57 | + | + | – | – | – | – | + | + |

*Recording electrode on diaphragm contralateral to nerve stimulated was near the midline.

+ = Action potentials present; – = Action potentials absent; RD = Right diaphragm; RC = Right crus; LD = Left diaphragm; LC = Left crus.

## DISCUSSION

These results are at variance with those previously published in that the muscle mass to the right of the esophagus was commonly innervated by both phrenic nerves. This difference with observations of others is perhaps not surprising, for the difficulties of the physiologic methods employed would appear fewer than those one must encounter in tracing nerve fibers down to their terminals in postmortem material. This finding is possibly all the more surprising because the inner margins of the esophageal hiatus are usually made up on both right and left by muscle bands which form the right crus, i.e., which insert to the right of the midline. One might assume, therefore, that branches of the right phrenic nerve would also pass to both sides of the esophagus and innervate the muscle adjacent to the esophagus. This, however, must be uncommon as compared to the types of innervation described in this and in the anatomic studies. To our knowledge, no instance has been described in which muscles to the left of the esophagus have been innervated by the right phrenic nerve.

The innervation and structure of the diaphragm have been recently reviewed by Botha[3] and by Ogawa[4] to whom the interested reader is referred. It may be observed in passing, however, that there are considerable variations in the position of the esophageal hiatus and in the origin and destination of the muscles which surround it. Some of these variations are doubtless predispositions to herniation. It is perhaps difficult to conceive that, where there is so much variability of structure, there should be the uniformity of innervation previously suggested.

## CONCLUSIONS

1. Innervation of the diaphragmatic crura was studied in 11 hepatectomized dogs by recording muscle action potentials from crura and diaphragms following phrenic nerve stimulation.

2. An action potential was always recorded from the diaphragm and corresponding crus when the ipsilateral phrenic nerve was stimulated.

3. In 4 of the dogs, the right crus was innervated also by the left or contralateral phrenic nerve. There was no instance in which the left crus was innervated by the right phrenic nerve.

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# EXPERIMENTAL USE OF NYLON MESH-SUPPORTED PERICARDIAL GRAFTS FOR CLOSURE OF FULL-THICKNESS ESOPHAGEAL DEFECTS

*Thomas C. Moore, M.D., and Gustavo Bounous, M.D.,*
*Indianapolis, Ind.*

THE availability in recent years of an increasing number of synthetic plastics which are well tolerated by the human body has stimulated considerable interest in the suitability of these materials for the support and replacement of certain anatomic structures. The success of synthetic vascular substitutes in arterial replacement and bypass and that of plastic meshes in the repair of hernial defects is well known.

The use of synthetic materials in esophageal surgery has been directed largely toward the use of impervious tubes for the replacement of resected segments of esophagus.[3, 4] There has been little interest in the usefulness of permeable plastics in esophageal surgery. The suitability of nylon mesh for the replacement of resected portions of esophageal musculature and the adequacy of its tolerance in this area has been reported recently by the authors.[6] In addition, the senior author[5] also has recorded the successful clinical use of a patch of nylon mesh for the support of a section of markedly weakened esophageal wall, following extramucosal resection of an obstructing subcarinal mediastinal granuloma. Only a tissue-paper thin mucosal remnant covered the esophageal lumen in the area of this resection.

While it is quite apparent that a permeable plastic such as nylon mesh could not be used alone for the closure of full-thickness esophageal defects, the experiences cited above have led us to investigate the feasibility of combining the use of nylon mesh for support and some nearby, relatively strong autogenous tissue for permanent or temporary water-tight closure of the defect. Pericardium was selected because of its availability and strength. The successful use of nylon net-supported tubes of autogenous pericardium in the experimental replacement of resected segments of thoracic aorta was described by Adler[2] in 1955. The combined use of nylon mesh and pericardium in esophageal surgery has not, to the authors' knowledge, been reported.

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From the Department of Surgery, Indiana University Medical Center, Indianapolis, Ind.
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## MATERIAL AND METHODS

Healthy mongrel dogs were used. An effort was made to limit the oral intake of liquids and solid food for a short period prior to operation. Intravenous Pentothal was employed for anesthesia. Oxygen was administered through an endotracheal tube by means of a mechanical respirator. The lower left pleural cavity was entered through an intercostal incision. The lower esophagus was mobilized and secured between Potts coarctation clamps. An oval full-thickness defect of standard size, measuring approximately 2.5 by 2 cm., was made by resecting a segment of esophageal wall (Fig. 1). The Potts coaretation clamps were useful not only in manipulation of the esophagus but also in the control of bleeding along the margins of the defect during graft placement and in prevention of possible contamination of the pleural cavity and the area of operation by gastric and upper esophageal contents.

A free pericardial graft was obtained by resecting a portion of the adjacent pericardium and tailoring it to the approximate size of the defect. The graft was secured to the margins of the defect with interrupted sutures of 5-0 arterial silk through the relatively thick and tough mucosal layer. Eight such anchoring sutures generally were employed. A continuous circumferential suture of 5-0 arterial silk then was used in an attempt to achieve a water-tight union between the pericardial graft and the mucosal layer at the margins of the defect. A piece of fine mesh nylon, of a type already shown to be tolerated well in the wall of the esophagus, was cut to the approximate size and shape of the defect. The nylon mesh fabric (241 F)* contained 270 by 204 threads per square inch. This supporting nylon mesh was secured to both esophageal mucosa and muscle layers at the margins of the defect with interrupted sutures of 5-0 arterial silk. The left thoracotomy incision was closed in layers and a catheter, left in place during the closure, was aspirated and withdrawn.

In a few animals, the use of the supporting nylon mesh was omitted so that these animals might serve as controls. In one animal, only nylon mesh was used to cover the defect.

The animals were given nothing by mouth for the first 24 hours after operation. On occasion, intravenous fluids were given during this period. For the following 10 days, they were given only milk by mouth. A soft meat diet then was given for one month prior to the resumption of a regular diet. Intramuscular penicillin, 200,000 units a day, was given for the first 5 days after operation.

## RESULTS

The 6 control animals in which a free pericardial graft was employed for water-tight esophageal defect closure without supporting nylon mesh all died 3 and 4 days following operation. In all 6, death was found to be due to perforation of the graft with gross contamination of the left pleural cavity. The one animal in which only nylon mesh was used for defect closure died of empyema 5 days after operation.

*Nylon fabric was obtained from Stehli Company Inc., 1372 Broadway, New York City.

A nylon mesh-supported free pericardial graft was used as described for closure of a standard full-thickness esophageal defect in 24 consecutive animals. Satisfactory defect closure with survival of the animal and no postmortem evidence of empyema was achieved in 21 of the 24. Three dogs died in 3, 5, and 6 days following operation, with empyema and evidence of disruption of the graft closure.

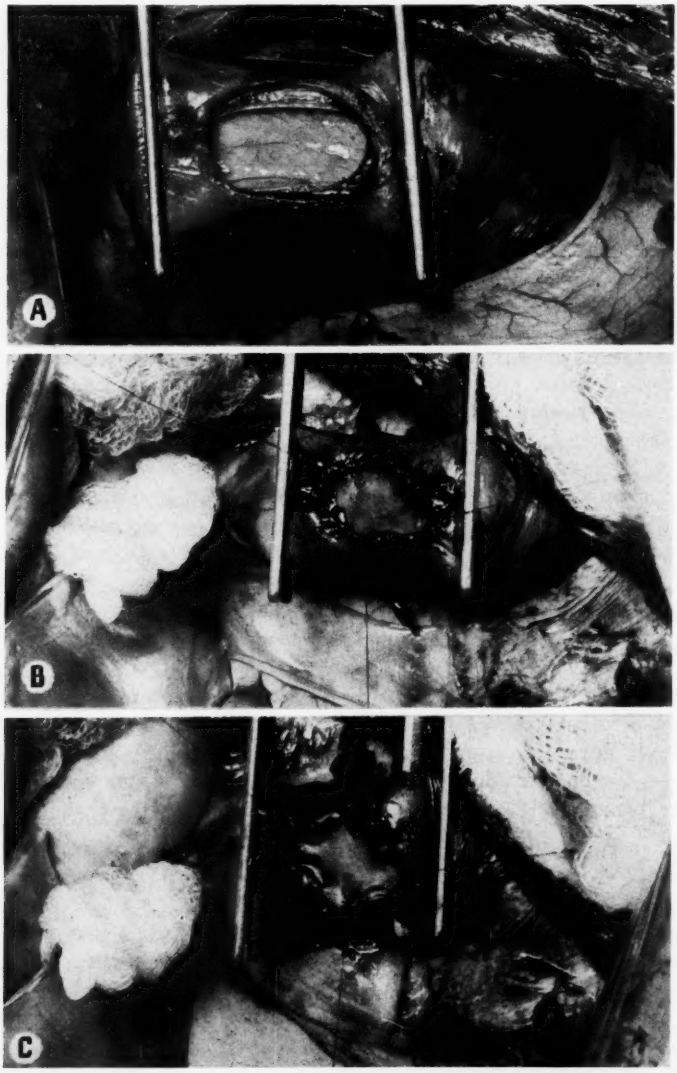


Fig. 1.—Photographs at the time of operation showing the standard full-thickness esophageal defect employed (*A*) before and (*B*) after closure with a free pericardial graft. Reinforcement of the free pericardial graft with fine mesh nylon is pictured in *C*.

The fate of the free pericardial graft and the supporting nylon mesh in surviving animals was of interest. The fate of these materials gave some insight into the pattern of esophageal defect healing. Animals were examined at autopsy at varying periods of time following operation in order to evaluate the integrity of the defect closure and to study the pattern of defect healing (Table I). The defect closure of pericardium and supporting nylon remained relatively intact until about 14 days after operation. At this point, the pericardial graft

TABLE I. SUMMARY OF POSTMORTEM FINDINGS IN THE 21 ANIMALS WITH SUCCESSFUL DEFECT CLOSURE

| DOG NO. | INTERVAL FROM OPERATION TO AUTOPSY (DAYS) | STATUS OF GRAFT AND DEFECT AREA AT AUTOPSY |
|---|---|---|
| 700 | 8 | Graft intact with nylon in place; no defect contracture; good closure |
| 658 | 9 | Graft intact with nylon in place; no defect contracture; no leak |
| 657 | 10 | Graft intact with nylon in place; no defect contracture; satisfactory closure |
| 662 | 11 | Graft intact with nylon in place; no leak or defect contracture |
| 708 | 14 | Graft intact; no contracture; nylon in place |
| 665 | 16 | Nylon loose in one area; defect closure adequate |
| 710 | 20 | Defect contracture; nylon starting to balloon into lumen of esophagus |
| 711 | 21 | Defect contracture; nylon ballooning into lumen; suture becoming free in one area |
| 701 | 27 | Defect contracture; nylon partially free and ballooning into lumen |
| 705 | 27 | Defect contracture; nylon ballooning into lumen |
| 706 | 27 | Defect contracture; nylon ballooning into lumen |
| 690 | 52 | Defect contracture; nylon ballooning into lumen and becoming detached |
| 688 | 55 | Defect contracture; nylon ballooning into lumen |
| 689 | 60 | Defect contracture; nylon ballooning into lumen |
| 677 | 63 | Nylon gone; some thinning of esophageal wall in area of contracted defect; defect closed by ingrowth of fibrous connective tissue |
| 692 | 68 | Nylon gone; defect contracted and closed by ingrowth of adjacent connective tissue |
| 666 | 76 | Defect contracture; nylon ballooning into lumen |
| 660 | 77 | Defect contracture; nylon ballooning into lumen and partially detached |
| 667 | 89 | Nylon gone; some granulation tissue projecting slightly into lumen in area of contracted defect |
| 656 | 137 | Nylon gone; defect contracted and closed by ingrowth of adjacent connective tissue |
| 638 | 161 | Nylon gone; defect contracted and closed by ingrowth of adjacent connective tissue; some thinning of esophageal wall in area of contracted defect |

tended to disappear, apparently sloughed into the lumen of the esophagus, and progressive defect contracture with ballooning of the nylon mesh patch into the lumen of the esophagus generally continued from 2 weeks to 2 months (Fig. 2). In 5 of the 7 animals examined more than 60 days after operation, the nylon mesh was no longer present and was presumed to have been discharged into the esophageal lumen. It was gone in all of 3 animals examined more than 80 days after operation. Although some thinning of the wall of the esophagus was noted

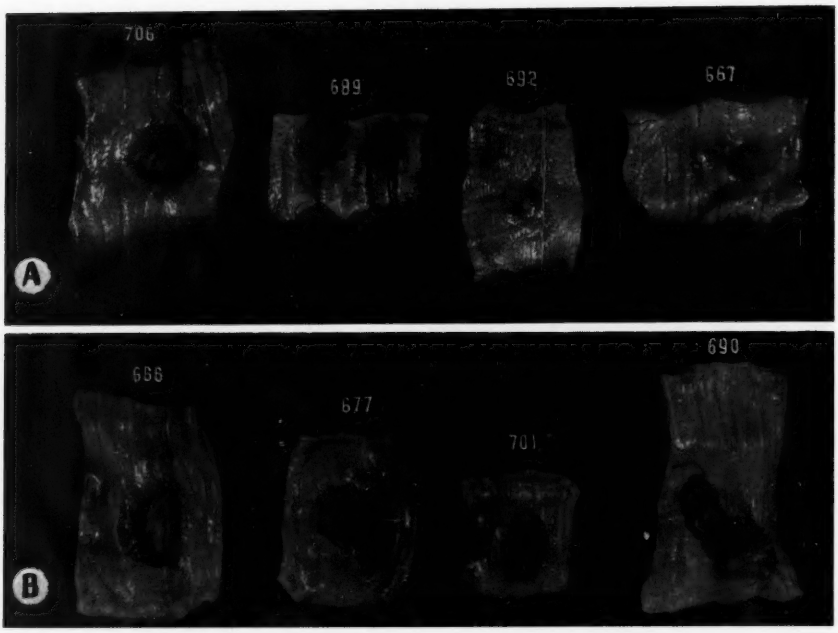


Fig. 2.—Photograph showing the appearance of the esophageal defect closure area from the lumen of the esophagus in 8 animals, on postmortem examination at varying periods following operation. In some, the nylon mesh has become partially detached or is ballooning into the lumen; in others it has disappeared. More detailed information on the specimens pictured may be obtained from Table I.

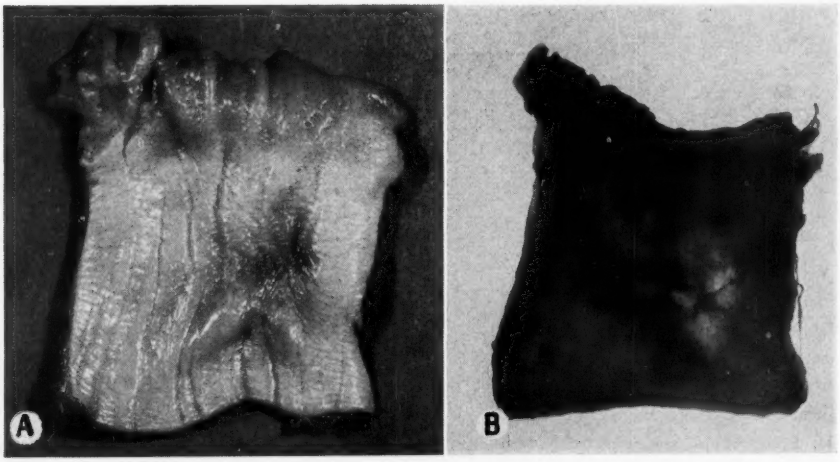


Fig. 3.—Photographs of the area of esophageal defect closure in Dog 638 over 5 months after operation. The healed area of defect closure may be seen in *A*. The thinning of the esophageal wall in the area of defect closure is demonstrated by transillumination in *B*.

in the area of defect closure after disappearance of the nylon (Fig. 3), no gross evidence of herniation or significant weakness of the esophageal wall in these areas was encountered. Furthermore, no contracture of the lumen of the esophagus at the level of the defect closure was found.

## DISCUSSION

The inadequacy of unsupported free pericardium for the closure of full-thickness esophageal defects of the standard size used in this study was conclusively demonstrated by the death of all 6 animals of graft disruption in this control group, from 3 to 4 days after operation. On the other hand, the survival of 21 of 24 dogs with successful defect closure when nylon mesh was added to support the free pericardial graft was regarded as significant evidence of the important role played by the supporting nylon in achieving successful defect closure.

When this study was begun, it was hoped that the free pericardial graft would survive and remain intact in the area of defect closure and that the nylon mesh would be incorporated into the newly formed esophageal wall through the ingrowth into its interstices of fibroblasts and blood vessels from surrounding connective tissue. This did not prove to be the case. The free graft of pericardium appeared to serve merely as a temporary seal until a permanent seal could develop from surrounding connective tissue. The pericardial graft generally disappeared in the early days after operation. Permanent defect closure was produced by a combination of defect contracture and healing in the area of permanent defect seal from surrounding connective tissue. The defect contracture caused a progressive ballooning of nylon mesh patch into the lumen of the esophagus. The nylon patch generally became detached from the margins of the contracted defect and disappeared approximately 2 months following operation.

Adler[1] in 1956 reported the results of experimental studies in which free grafts of pericardium were used for the support of purposely faulty end-to-end esophageal anastomoses. The pericardial graft was secured to the esophagus in the area to be protected with a thrombin-fibrinogen coagulum. He found that the free pericardial graft secured in this manner protected the faulty anastomoses in 5 of 10 dogs. Only 2 of 10 dogs with unprotected faulty anastomoses survived and one of these dogs developed a localized abscess.

The greater success of nylon mesh-supported pericardial grafts, as experienced in this study, would appear to suggest that this method of closure of esophageal defects of limited size and of reinforcement for questionable esophageal anastomoses is worthy of a clinical trial.

## SUMMARY

The experimental use of nylon mesh-supported free pericardial grafts for the closure of relatively large full-thickness esophageal defects of standard size is reported. The operation was carried out as described in 24 dogs. Twenty-one of the animals survived with successful defect closure. All of 6 dogs in

which only a free pericardial graft was used to cover the defect died of graft disruption and gross pleural contamination 3 and 4 days after operation. The pattern of defect healing is described and possible clinical application of these observations is discussed.

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# FOREIGN BODY BRONCHOESOPHAGEAL FISTULA*

*H. V. Swindell, Major, USAF(MC),** Thomas Holcomb, Major, USAF(MC),*** and Daniel C. Campbell, Lieutenant Colonel, USAF(MC),** Lackland Air Force Base, Texas*

BRONCHOESOPHAGEAL fistulas due to esophageal foreign bodies are seldom reported. A review of the medical literature through 1957 yielded only 16 cases, 11 of which were concentrated in the doctorate thesis of Cirot published in 1898 at Lyons, France. Coleman and Bunch[1] in a collective review of the literature through 1950 added 4 cases, one, however, due to tracheal rather than esophageal foreign body. In 1957, 2 additional cases were reported by Pouyanne and his associates[5] and Lemariey,[4] both occurring in nursing infants.

Typically, the symptoms are the same as for congenital tracheoesophageal fistulas, with the addition of a history of esophageal foreign body ingestion. The classic symptom is severe cough following the swallowing of liquid or occasionally of solid food. This cough can sometimes be controlled by positioning the patient while swallowing. The appearance of râles in one or both lung fields following the ingestion of liquids (sign of Ono) is described as pathognomonic of tracheoesophageal communication. Persistent abdominal distention is common due to the continuous passage of air from the tracheobronchial tree into the esophagus. Frequently atelectasis and pneumonia occur if the fistula has been present for any length of time.

Unfortunately, as demonstrated in our case, any of the symptoms, except the pulmonary complications, may be absent or so minimal that they are completely overshadowed by the pulmonary findings. This is particularly true when the foreign body is nonopaque and no history of ingestion obtained.

## CASE REPORT

J. B., No. 294779, a 2-year-old white female, had been followed in the Pediatric Clinic for approximately one year for asthma and recurrent respiratory infections. Acutely ill, with left lower lobe "pneumonia" and a left pleural effusion, she was admitted to the Pediatric Service on June 25, 1958. Thoracentesis yielded a serosanguineous fluid. Culture and cell

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*This paper represents the personal viewpoint of the author and is not to be construed as a statement of official Air Force policy.

**From Department of Surgery, USAF Hospital, Lackland AFB, Texas; Don S. Wenger, Colonel, USAF(MC), Chairman.

***Chief, Pediatric Service, USAF Hospital, Lackland AFB, Texas.

blocks were negative. Repeated x-ray studies revealed left lower lobe atelectasis, and bronchoscopy was performed. The left main-stem bronchus and the lower lobe bronchus were edematous, and injected. Serosanguineous fluid was aspirated from the bronchus, and a beta hemolytic streptococcus was cultured. The effusion and pneumonia cleared rapidly on treatment with penicillin. The left lower lobe atelectasis persisted, but finally improved on positive pressure breathing with Alevaire and Isuprel. The patient was discharged after 28 days. Intermittent positive pressure therapy was continued on an outpatient basis. The patient was asymptomatic on this regimen for one month. During a routine weekly check in the Pediatric Clinic, the patient was found to have a recurrence of the complete atelectasis of the left lung. She was immediately readmitted on Aug. 22, 1958.

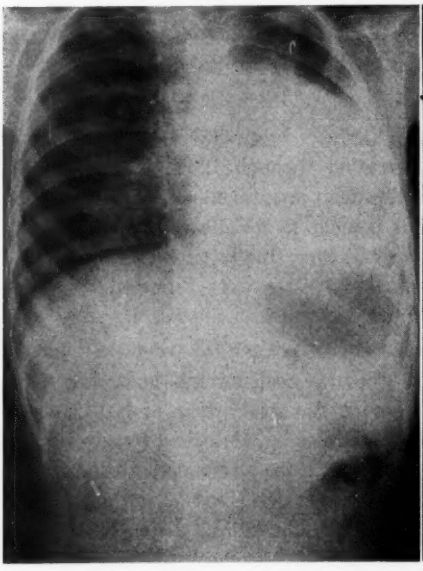

Fig. 1.

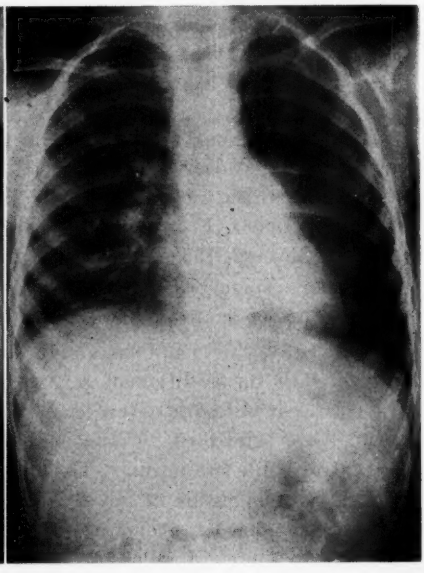

Fig. 2.

Fig. 1.—Chest roentgenogram at time of first admission showing left pleural effusion.
Fig. 2.—Chest roentgenogram at time of discharge from first admission showing completely clear left chest.

The patient was not clinically ill. The positive physical signs were limited to the chest and revealed a respiratory lag on the left with diminished breath sounds. Complete blood count and urinalysis were within normal limits. Stool examinations revealed normal trypsin digestion.

The patient underwent bronchoscopy immediately after admission. The examination was unsatisfactory due to mechanical difficulties. She was placed in moist oxygen with Isuprel and Alevaire. On the following day, x-ray studies revealed almost complete clearing of the atelectasis. Twelve days later chest roentgenograms again revealed complete left lower lobe collapse and bronchoscopy was again performed. Considerable secretions were aspirated from the main-stem bronchus. The upper lobe orifice was patent. Just below the upper lobe takeoff, the bronchus was obstructed by a reddish friable mass which was removed by biopsy forceps. The lower lobe bronchus was then aspirated and noted to exchange air adequately. Prophylactic tracheotomy was performed. The pathologic report of the biopsy specimen was granulation tissue consistent with foreign body reaction. The patient's clinical condition began to deteriorate. Gradually she became more apathetic and depressed. The temperature

rose daily to 102° F. and penicillin therapy was supplemented by Chloromycetin and erythromycin. Serial chest roentgenograms revealed varying degrees of aeration of the left lower lobe despite the septic course.

On Sept. 24, 1958 (20 days after bronchoscopy), a left lower lobe lobectomy was performed. A nonopaque foreign body of the bronchus was considered the most likely etiologic agent and in view of x-ray evidence of possible abscess formation and progressive deterioration of the patient despite massive antibiotics, operation was advised.

The operation was carried out under hypothermia with reduction of body temperature to 99° R. At operation, the left lower lobe was completely consolidated and massive inflammatory adenopathy was present in the hilum. On sectioning the bronchus, a copious amount of thick purulent secretion was aspirated from both the main-stem and upper lobe bronchi. The upper lobe expanded well after this. The patient withstood the operation amazingly well. The specimen grossly had a peculiar polypoid appearance, and a tentative frozen section diagnosis of papillomata of the bronchus was made.

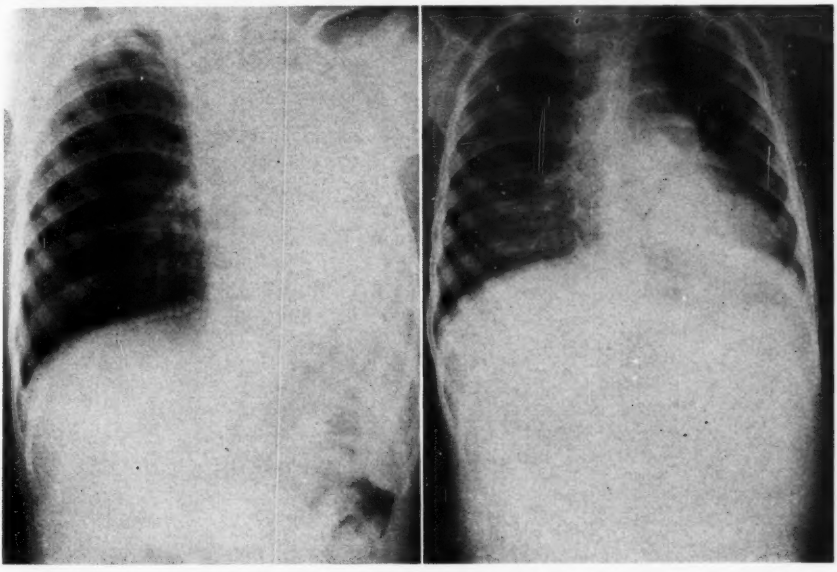

Fig. 3. Fig. 4.

Fig. 3.—Chest roentgenogram on second admission showing total atelectasis of left lung.
Fig. 4.—Chest roentgenogram immediately preoperative, showing residual left lower lobe atelectasis and possible abscess.

Hypothermia was maintained for 24 hours postoperatively and then discontinued. The patient did well until noon of the second postoperative day when she exhibited severe respiratory distress and a temperature of 104° F. Abdominal distention was first noted at this time. Intratracheal and gastric suction were employed with some relief of symptoms. The patient appeared to be improving when she suddenly became cyanotic, gasped for breath, and died.

The pertinent autopsy findings were limited to the chest. The right lung was expanded, crepitant, and normal. The left pleural space contained a minimal amount of fluid. The left upper lobe was expanded, but indurated. The entire thoracic contents were removed en bloc. The esophagus was opened longitudinally, and in the middle one third there was a 2 by 2 cm. plastic foreign body wedged across the lumen. The edges were sharp and

irregular. One corner of the foreign body extended through a slit-like fistula, 0.5 cm. in length, which communicated with the left main-stem bronchus at the level of the upper lobe orifice. The fistulous tract was fibrous and epithelized. The lobectomy stump was well sealed. The remainder of the autopsy was within normal limits.

Following autopsy, a careful search of old outpatient records, and detailed questioning of the parents, revealed that the patient had swallowed a plastic-covered button from her mother's nurse uniform in April, 1957 (18 months prior to surgery). She regurgitated the button 5 days after ingestion and was apparently normal. No other history of foreign body ingestion could be obtained.

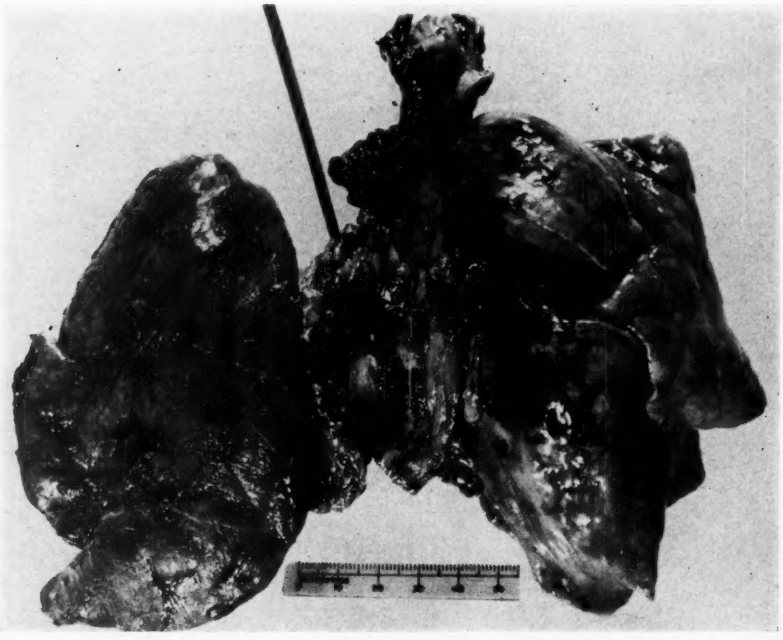

Fig. 5.—Autopsy specimen viewed posteriorly showing foreign body in esophagus. A metal probe has been inserted through the fistula.

## DISCUSSION

This case illustrates the vague or practically nonexistent symptomatology that may be presented by a bronchoesophageal fistula secondary to a retained esophageal foreign body. The only symptoms noted during the 18-month period were two transient episodes of wheezing ascribed to asthma. Never was there x-ray evidence to suggest the presence of a foreign body. However, in retrospect, the entire clinical course becomes logical. One can only suppose that the plastic covering of the button became wedged in the esophagus in April, 1957, and that the patient regurgitated only the metal frame 5 days later, a fact not noted by the parents. Six months later a diagnosis of asthma was made because of wheezing.

It is easy, in retrospect, to visualize the irritative process that produced the wheezes. For a period of over a year, episodes of respiratory infection increased in number, followed finally by pneumonia and atelectasis as the fistula increased in size. The most confusing part of the picture was the absence of strangulation symptoms on liquid ingestion. We feel that this was due to the impaction of the foreign body in the fistula which prevented the dumping of swallowed fluids into the tracheobronchial tree.

One wonders how the diagnosis could have been made antemortem. The occurrence of "asthma" in a young child previously free of allergic symptoms certainly should suggest the possibility of foreign body ingestion. Critical review of the case has failed to reveal any symptoms which would have indicated the diagnosis. At the time of lobectomy, the fistula was above the level of bronchial amputation and not visualized. At bronchoscopies, it was obscured by edema, granulation tissue, and by the fact that the foreign body did not actually protrude into the bronchus. The only means, other than esophagoscopy, by which diagnosis could possibly have been made would have been by means of a Lipiodol swallow.

SUMMARY

A case of bronchoesophageal fistula due to ingestion of foreign body is reported with a review of 16 cases from world literature. Foreign body ingestion must be considered when signs of asthma appear in a previously allergy-free patient. Examination of the esophagus should be considered in cases of unexplained recurrent pneumonia and atelectasis.

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# AN ELECTRICALLY CONDUCTIVE CARDIAC SUTURE

*Edward A. Fitch, M.D.,* and Charles P. Bailey, M.D.,***
*Philadelphia, Pa.*

THE necessity for applying an artificial stimulus to the heart to evoke effective ventricular contractions arises occasionally. This problem is of increasing frequency of occurrence, in part due to the development of "open-heart" surgery with its incidence of surgical interruption of the conduction system. Usually these incidences occur at critical times, and the patient's survival depends on the efficacy of the artificially stimulated contractions.

This résumé is confined to the discussion of those syndromes in which ventricular myocardial contractility is adequate and the primary defect is the absence of a stimulus of sufficient frequency and or/intensity.

An intermittent electrical impulse is the most reliable type of artificial stimulus. Several "pacemakers" are commercially available. The electrodes can be applied externally at two points on the chest wall and the stimulus increased in intensity until a perceptible pulse, synchronous with the pacemaker rhythm, indicates that the ventricles are responding.

Electrodes can be applied internally via a thoracotomy incision, and the response further evaluated by direct vision.

There are several difficulties. The external electrodes cause spasmodic contractions of all skeletal muscle within their field, as well as myocardial tissue. The resultant artificial clonic local convulsion soon becomes intolerable to the conscious patient, and severely handicaps the observation and examination of the critical patient.

When peripheral vascular hypo- or hypertonia renders pulses imperceptible, the effectiveness of the electrical stimulus is open to question. With a thready pulse and jerking chest musculature, one is never sure (in the closed chest) at what point an increasing intensity is sufficient to stimulate the myocardium.

It is suggested that in medical heart block the contractions be re-established by use of a pacemaker and external electrodes. If tentative cessation of the stimulation results in immediate failure of the circulation, the chest should be opened.

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*Public Health Service Research Fellow of the National Heart Institute. Resident in Thoracic Surgery, Hahnemann Medical College and Hospital, Philadelphia, Pa.

**Director of Bailey Thoracic Clinic, Philadelphia, Pa.

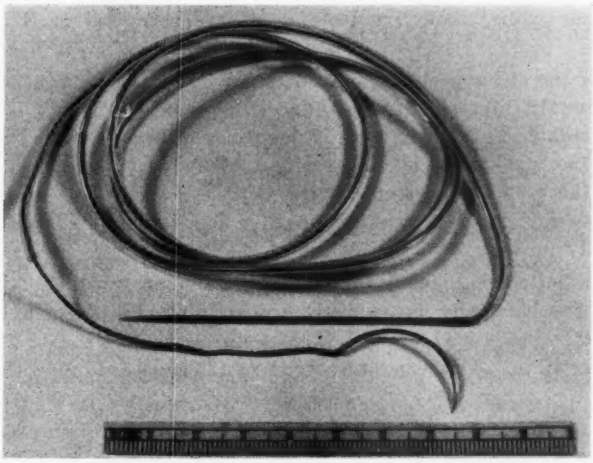

Fig. 1.—The electrically conductive suture is commercially available in sterile packets. Silver wire is braided around a silk core. Polyvinyl tubular insulation allows autoclaving.

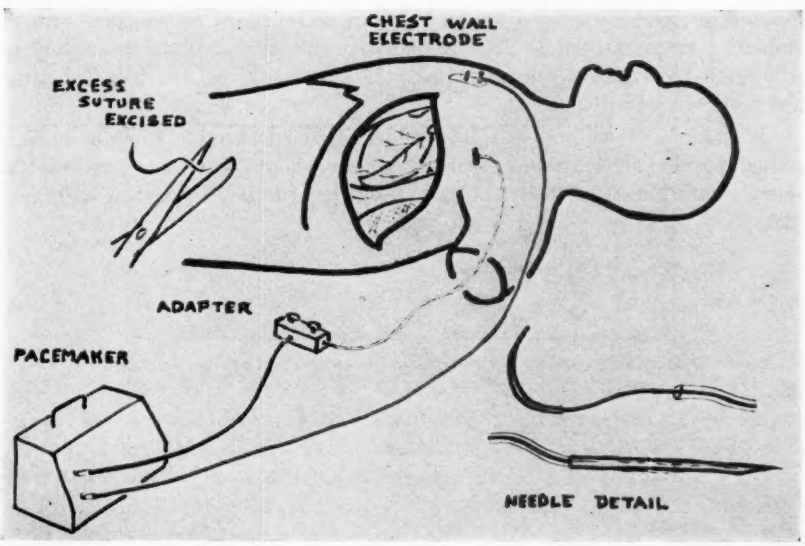


Fig. 2.—Use of the electrode wire sutured into the myocardium with a nonspecific external electrode.

An opened chest facilitates evaluation of the heartbeat by direct vision. However, it is axiomatic that the sooner the wound is closed, the better. Closure of the incision while the external electrodes are in operation is clumsy and difficult.

Many of the above problems have been solved by the application of a wire electrode directly into the myocardium. Such an apparatus is now commercially available.* A silver wire is doubly armed with swaged needles, one curved and one straight. The entire wire is insulated with a polyvinyl sheath, except for the 4 inches adjacent to the curved needle The surrounding insulation is swaged into the straight needle. The suture is supplied in sterile packets ready for use.

A single stitch is taken into the ventricular myocardium. The curved needle and the bare wire are pulled through until the polyvinyl insulation encounters the muscle. The needle is then cut off leaving a 1 to 2 cm. length of wire protruding. This can be secured to the myocardium with a 5-0 silk stitch.

A spot on the chest wall is then chosen for the penetration of the straight needle. This should be away from the incisional edges so it will not be dislodged during wound closure. It should be in such a location that the suture, when withdrawn from the chest, will pull out of the heart in the long axis of the wire, and not lacerate the myocardium by sawing action as in a right angle pull-out. The straight needle is attched directly to the pacemaker attachment. Adapters are commercially available* to join all standard pacemakers with the conductive straight needle. The second (nonspecific) electrode is applied externally to the right of the sternum or, as an alternative, two internal suture electrodes can be utilized.

With such a setup, one can be confident that an effective stimulus is being applied to the myocardium. This assurance should eliminate one variable from the multifaceted problem of sustaining the critically ill patient with heart block.

*Ohio Chemical Co., Madison, Wis.

# THREE USEFUL CLAMPS FOR CARDIOVASCULAR SURGERY

*S. Abraham Cuadros, M.D., Lima, Peru*

IT IS CLEAR that cardiovascular surgery has progressed at an ever accelerating rate in the last few years, providing in a short time the answers to a large number of problems which for a long time remained insoluble. To keep pace with expanding horizons, continued advances in surgery, and new and perfected techniques, it is necessary to use new and better instruments.

One cannot fail to recognize that one of the most ingenious and useful instruments in this field of surgery is the Potts-Smith clamp, with its advantages of safety for partial occlusion of the vessel lumen. It has, however, the disadvantage that it cannot always be applied as desired, for instance when full mobilization of a vessel is difficult, or when there are important branches at the exact site of the clamping which cannot be divided, and also when it is necessary to apply it rapidly in situations of emergency. For these reasons, Blalock introduced an improved modification; yet even this has not proved fully satisfactory.

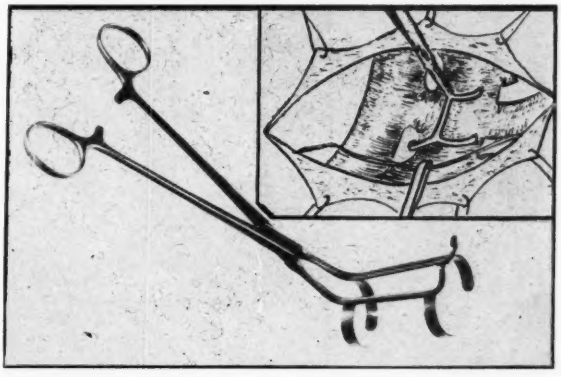

Fig. 1.—This figure shows the first clamp and one use of this clamp in an atypical patient ductus arteriosus which is just below the carotid artery.

To combat the difficulties just mentioned, two clamps have been devised. The first clamp (Fig. 1) has two metal arcs on each limb. When the clamp is closed these form a circle which embraces the vessel to be occluded. With this, one avoids all the disadvantages of the Potts-Smith clamp. The drawing in Fig. 1 illustrates the clamp in use.

The clamp seen in Fig. 2 has two small rings on each limb. This allows a ligature to be passed around the vessel and fixes it safely, giving the clamp

From the Karolinska Institute, Stockholm, Sweden.
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an extremely simple and effective character, as can be seen in the drawing in Fig. 2.

One of the difficulties often encountered in the clamping of the aorta in its approach is the slipping of the end of the clamp, due to aortic arterial pressure and, in certain circumstances, this can become a serious problem. Having

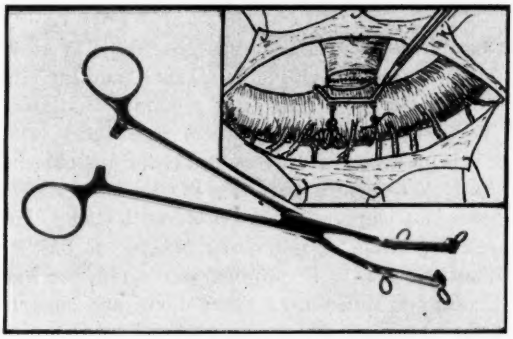

Fig. 2.—This figure shows the second clamp and one use of this clamp in a Potts operation without cutting the branches of the descending aorta.

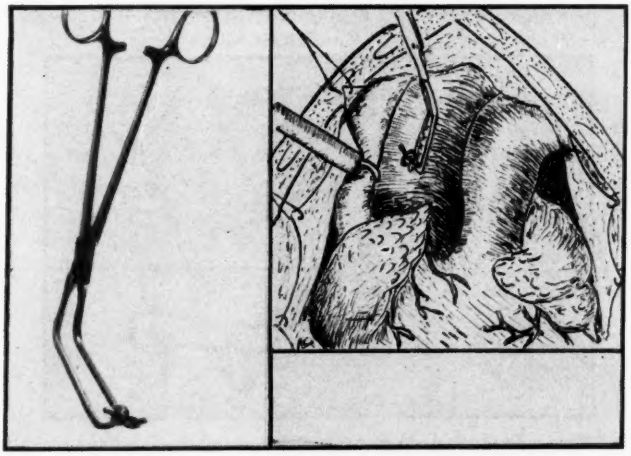

Fig. 3.—This figure shows the third clamp and its use in aortic valvulotomy under direct vision (hypothermia).

noticed that this slipping is due to weakness of the clamp at its free extremity, it has been found possible to overcome this difficulty with a hinged screw which, when screwed up, effectively prevents the clamp from slipping at its free end. A third clamp, incorporating this hinged screw, can be seen in Fig. 3. The clamp in use is illustrated in the drawing in Fig. 3.

I wish to thank Prof. A. L. d'Abreu from the Queen Elizabeth Hospital (University of Birmingham, England) in whose Laboratory of Experimental Surgery these instruments were devised.

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BAILEY, CHARLES P.---------- 249 N. Broad St., Philadelphia 7, Pa.
BARKLEY, HOWARD T.---------- 4414 Montrose Blvd., Houston 6, Texas
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BEATTIE, EDWARD J., JR.---------- 1753 W. Congress Parkway, Chicago 12, Ill.
BEECHER, HENRY K.---------- Massachusetts General Hospital, Boston 14, Mass.
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BORTONE, FRANK---------- 2765 Hudson Blvd., Jersey City 6, N. J.

*If any corrections should be made in the spelling of names or in the addresses of members, please notify the Administrative Assistant of the Association, Miss Ada Hanvey, 308 Carondelet Building, 7730 Carondelet Avenue, St. Louis 5, Mo.

BOSHER, LEWIS H.----1200 E. Broad St., Richmond 19, Va.
BOYD, DAVID P.----Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
BRADSHAW, HOWARD H.----Bowman Gray School of Medicine, Winston-Salem, N. C.
BRANTIGAN, OTTO C.----104 W. Madison St., Baltimore 1, Md.
BREWER, LYMAN A., III----2010 Wilshire Blvd., Los Angeles 57, Calif.
BRINDLEY, GEORGE V., JR.----Scott and White Clinic, Temple, Texas
BROWN, ROBERT K.----1624 Gilpin St., Denver 6, Colo.
BROWNRIGG, GARRETT M.----47 Queens Road, St. Johns, Newfoundland
BRUNEAU, JACQUES----847 Rue Cherrier, Montreal 24, Canada
BUCKINGHAM, WILLIAM W.----314 Professional Bldg., Kansas City 6, Mo.
BUGDEN, WALTER F.----Medical Arts Bldg., Syracuse 10, N. Y.
BURFORD, THOMAS H.----Barnes Hospital, St. Louis 10, Mo.
BYRON, FRANCIS X.----120 S. Lasky Dr., Suite 203, Beverly Hills, Calif.

CARLSON, ROBERT I.----1410 West Michigan St., Indianapolis 7, Ind.
CARR, DUANE----20 S. Dudley St., Memphis 3, Tenn.
CARTER, MAX G.----670 George St., New Haven, Conn.
CHAMBERLAIN, JOHN MAXWELL----23 East 79th St., New York 21, N. Y.
CHAMBERS, JOHN S., JR.----2850 Sixth St., San Diego 3, Calif.
CHESNEY, JOHN G.----2300 Biscayne Blvd., Miami 37, Fla.
CLAGETT, O. T.----Mayo Clinic, Rochester, Minn.
CLATWORTHY, H. WM., JR.--Children's Hospital, 17th St. at Livingston Park, Columbus 5, Ohio
CLOWES, GEORGE H. A., JR.----3395 Scranton Road, Cleveland 9, Ohio
COHN, ROY B.----Stanford University Hospital, San Francisco 15, Calif.
COLEMAN, FRANK P.----1111 W. Franklin St., Richmond 20, Va.
CONDON, WILLIAM B.----1104 Republic Bldg., Denver 2, Colo.
CONKLIN, WILLIAM S.----1016 Standard Ins. Bldg., Portland 5, Ore.
COOLEY, DENTON A.----Baylor University College of Medicine, Houston 25, Texas
COTTON, BERT H.----1321 N. Vermont Ave., Los Angeles 27, Calif.
CRANDELL, WALTER B.----Veterans Adm. Hospital, White River Junction, Vt.
CREECH, OSCAR, JR.----Tulane University School of Medicine, New Orleans 12, La.
CRIMM, PAUL D.----Boehne Hospital, Evansville 12, Ind.
CROSS, FREDERICK S.----11311 Shaker Blvd., Cleveland 4, Ohio
CURRERI, ANTHONY R.----1300 University Ave., Madison 6, Wis.
CUTLER, PRESTON R.----535 East 1st South, Salt Lake City 2, Utah

DAILEY, JAMES E.----4109 Montrose Blvd., Houston 6, Texas
DANIEL, ROLLIN A.----410 Medical Arts Bldg., Nashville 12, Tenn.
DANIELS, ALBERT C.----490 Post St., San Francisco 2, Calif.
DAUGHTRY, DEWITT C.----4201 Lake Road, Bay Point, Miami 37, Fla.
DAVIS, EDGAR W.----1150 Connecticut Ave., Washington 6, D. C.
DAY, J. CLAUDE----307 David Whitney Bldg., Detroit 26, Mich.
DE BAKEY, MICHAEL E.----Baylor University, Dept. of Surgery, Houston, Texas
DECAMP, PAUL T.----3503 Prytania Street, New Orleans 15, La.
DELARUE, NORMAN C.----25 Donlea Drive, Toronto 17, Ont.
DENNIS, CLARENCE----989 Edgewood Ave., Pelham Manor, N. Y.
DESHAIES, GEORGES----37 Bellingham Road, Montreal, Que.
DETERLING, RALPH A.----180 Ft. Washington Ave., New York 32, N. Y.
DODRILL, FOREST D.----621 David Whitney Bldg., Detroit 26, Mich.
DOMM, SHELDON E.----1918 W. Clinch Ave., Knoxville 16, Tenn.
DORNER, RALPH A.----710 Equitable Bldg., Des Moines 9, Iowa
DORSEY, JOHN M.----636 Church St., Evanston, Ill.
DRAKE, EMERSON H.----18 Bramhall St., Portland 3, Maine
DRASH, EVERETT C.----University of Virginia Hospital, Charlottesville, Va.
DUGAN, DAVID J.----459 30th St., Oakland 9, Calif.

EFFLER, DONALD B.---Euclid and East 93rd Sts., Cleveland 6, Ohio
EHRENHAFT, JOHANN L.---University of Iowa, Iowa City, Iowa
ELLIS, F. HENRY, JR.---Mayo Clinic, Rochester, Minn.
ELLISON, ROBERT G.---Medical College of Georgia, Augusta, Ga.
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Box 73, Letterman Army Hospital, San Franciso, Calif.

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GALE, JOSEPH W.---Wisconsin General Hospital, Madison 6, Wis.
GEARY, PAUL---909 Park Ave., Plainfield, N. J.
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GERBODE, FRANK---Stanford University Hospital, San Francisco 15, Calif.
GIBBON, JOHN H., JR.---1025 Walnut St., Philadelphia 7, Pa.
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HARDY, JAMES D.---University of Mississippi Medical Center, Jackson, Miss.
HARKEN, DWIGHT E.---67 Bay State Road, Boston 15, Mass.
HARPER, FREDERICK R.---1104 Republic Bldg., Denver 2, Colo.
HARRISON, ALBERT W.---Medical Branch, Univ. of Texas, Galveston, Texas
HARRISON, ELLIOTT---1862 West Broadway, Vancouver 9, B. C.
HARTER, JOHN S.---212 Brown Bldg., Louisville 2, Ky.
HELMSWORTH, JAMES A.---Cincinnati General Hospital, Cincinnati 29, Ohio
HIGGINSON, JOHN F.---1430 Chapala St., Santa Barbara, Calif.
HIMMELSTEIN, AARON---70 East 96th St., New York 28, N. Y.
HOCHBERG, LEW A.---135 Eastern Parkway, Brooklyn 38, N. Y.
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HOPKINS, WILLIAM A.---1293 Peachtree St., N.E., Atlanta 9, Ga.

HUDSON, THEODORE R.---55 E. Washington St., Chicago 2, Ill.
HUGHES, FELIX A., JR.---Kennedy Hospital, Memphis 17, Tenn.
HUMPHREYS, GEORGE H., II---180 Fort Washington Ave., New York 32, N. Y.
HURLEY, GERARD A. P.---1538 Sherbrooke St., W., Montreal, Que.
HURWITT, ELLIOTT S.---Montefiore Hospital, New York 67, N. Y.
HURWITZ, ALFRED---Maimonides Hosp., 4802 Tenth Ave., Brooklyn 19, N. Y.

JANES, ERNEST C.---250 Main St., East, Hamilton, Ont.
JARVIS, FRED J.---1115 Columbia St., Seattle 4, Wash.
JOHNSON, ELGIE K.---230 Hilton St., Hempstead, N. Y.
JOHNSON, JULIAN---3400 Spruce St., Philadelphia 4, Pa.
JOHNSTON, JAMES H., JR.---710 N. State St., Jackson 2, Miss.
JONES, JOHN C.---1136 West 6th St., Los Angeles 17, Calif.
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JULIAN, ORMAND C.---25 E. Washington St., Chicago 2, Ill.

KAY, EARLE B.---10465 Carnegie Ave., Cleveland 6, Ohio
KEELEY, JOHN L.---30 North Michigan Ave., Chicago 2, Ill.
KELLEY, WINFIELD O.---Uncas-on-Thames, Norwich, Conn.
KENT, EDWARD M.---3500 Fifth Ave., Pittsburgh 13, Pa.
KERGIN, FREDERICK G.---Toronto General Hospital, Toronto 2, Ont.
KESSLER, CHARLES R.---1321 21st Way, South, Birmingham 5, Ala.
KING, RICHARD---Suite 233, 340 Boulevard, N.E., Atlanta 12, Ga.
KINSELLA, THOMAS J.---1251 Medical Arts Bldg., Minneapolis 2, Minn.
KIRBY, CHARLES K.---3400 Spruce Street, Philadelphia 4, Pa.
KIRKLIN, JOHN W.---Mayo Clinic, Rochester, Minn.
KIRSCHNER, PAUL A.---2 East 92nd St., New York 28, N. Y.
KITTLE, C. FREDERICK---University of Kansas Medical Center, Kansas City 12, Kan.
KLASSEN, KARL P.---Ohio State University, Columbus 15, Ohio
KLEPSER, ROY G.---1835 Eye St., N.W., Washington 6, D. C.
KLOPSTOCK, ROBERT---Veterans Adm. Hospital, Brooklyn 9, N. Y.

LAIRD, ROBERT---399 Bathurst St., Toronto, Ont.
LAM, CONRAD R.---Henry Ford Hospital, Detroit 2, Mich.
LAMBERT, ADRIAN---768 Park Ave., New York 21, N. Y.
LAMPSON, R. STARR---85 Jefferson St., Hartford 14, Conn.
LANGSTON, HIRAM T.---1919 West Taylor St., Chicago 12, Ill.
LAUREY, JAMES R.---5710 16th St., N.W., Washington 11, D. C.
LEEDS, SANFORD E.---2211 Post St., San Francisco 15, Calif.
LEES, WILLIAM M.---7000 N. Kenton Ave., Lincolnwood 46, Ill.
LEWIS, F. JOHN---Northwestern Univ. Med. School, Chicago 11, Ill.
LILLEHEI, C. WALTON---University Hospitals, Minneapolis 14, Minn.
LINDSKOG, GUSTAF E.---50 Marvel Road, New Haven, Conn.
LONGMIRE, WILLIAM P., JR.---UCLA School of Medicine, Los Angeles 24, Calif.
LYNCH, JOSEPH P.---1180 Beacon St., Brookline 46, Mass.

MACKLER, S. ALLEN---104 S. Michigan Ave., Chicago 3, Ill.
MACMANUS, JOSEPH---491 Delaware, Buffalo 2, N. Y.
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MAJOR, ROBERT C.---842 Greene St., Augusta, Ga.
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MAURER, ELMER P. R.---827 Union Central Bldg., Cincinnati 2, Ohio
MAUTZ, F. R.---10515 Carnegie Ave., Cleveland 6, Ohio
MAYER, JOHN H., JR.---503 Plaza Parkway Bldg., Kansas City 12, Mo.
MCDONALD, JOHN R.---Harper Hosp., 3825 Brush St., Detroit, Mich.
MCINTOSH, CLARENCE A.---1390 Sherbrooke St., W., Montreal, Que.
MELTZER, HERBERT---505 Medical Arts Bldg., Edmonton, Alberta, Canada
MENDELSOHN, HARVEY J.---2065 Adelbert Road, Cleveland 6, Ohio
MERENDINO, K. ALVIN---University of Washington, Seattle 5, Wash.
MERKEL, CARL G.---8 Church St., Saranac Lake, N. Y.
MEYER, BERTRAND W.---922 Keatley Road, La Canada, Calif.
MILLS, WALDO O.---1445 Medical and Dental Bldg., Seattle 1, Wash.
MINOR, GEORGE R.---Univ. of Virginia Hospital, Charlottesville, Va.
MISCALL, LAURENCE---11 East 68th St., New York, N. Y.
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MULLER, WM. H., JR.---Univ. of Virginia Hospital, Charlottesville, Va.
MULVIHILL, DANIEL A.---15 East 77th St., New York 21, N. Y.

NEMIR, PAUL, JR.---University of Pennsylvania, Philadelphia 4, Pa.
NEPTUNE, WILFORD B.---135 Francis St., Boston 15, Mass.
NEWMAN, MELVIN M.---450 Clarkson Ave., Brooklyn 3, N. Y.

OATWAY, WILLIAM H., JR.---La Vina Sanatorium, Altadena, Calif.
OLSEN, ARTHUR M.---102 2nd Ave., S.W., Rochester, Minn.
O'NEILL, THOMAS J. E.---Suite 110 Centennial Bldg., Philadelphia 25. Pa.
O'ROURKE, PAUL V.---307 David Whitney Bldg., Detroit 26, Mich.
OVERHOLT, RICHARD H.---135 Francis St., Boston 15, Mass.

PAINE, JOHN R.---The Buffalo General Hosp., 100 High St., Buffalo 14, N. Y.
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ROGERS, W. L.---490 Post St., San Francisco 2, Calif.
ROSEMOND, GEORGE P.---3401 Broad St., N., Philadelphia 40, Pa.
RUMEL, WILLIAM R.---535 East 1st South, Salt Lake City 2, Utah

SALYER, JOHN M.---Box 6388, Fitzsimons Hosp., Denver 8, Colo.
SAMSON, PAUL C.---3959 Happy Valley Road, Lafayette, Calif.
SANGER, PAUL W.---Doctors Bldg., Kings Drive, Charlotte, N. C.
SAROT, IRVING A.---111 East 69th St., New York 21, N. Y.
SCANNELL, J. GORDON---Massachusetts General Hospital, Boston 14, Mass.
SCHAFFNER, VERNON D.---12 Cornwallis St., Kentville, Nova Scotia
SCHMIDT, HERBERT WM.---Mayo Clinic, Rochester, Minn.
SCOTT, HENRY W., JR.---Vanderbilt University Hospital, Nashville 5, Tenn.
SEALY, WILL C.---Duke University Hospital, Durham, N. C.
SEILER, HAWLEY H.---517 Bayshore Blvd., Tampa 6, Fla.
SELEY, GABRIEL P.---540 Park Ave., New York 21, N. Y.
SHAW, ROBERT R.---3810 Swiss Ave., Dallas, Texas
SHEFTS, LAURENCE M.---614 Medical Professional Bldg., San Antonio 12, Texas
SHUMACKER, HARRIS B., JR.---Indiana Univ. Medical Center, Indianapolis 7, Ind.
SKINNER, EDWARD F.---20 S. Dudley St., Memphis 3, Tenn.
SKINNER, GEORGE F.---36 Coburg St., St. Johns, N. B.
SLOAN, HERBERT---University Hospital, Ann Arbor, Mich.
SNYDER, JOHN M.---1236 Moffitt Ave., Bethlehem, Pa.
SOMMER, GEORGE N. J., JR.---120 W. State St., Trenton 8, N. J.
SOUTTER, LAMAR---80 East Concord St., Boston 18, Mass.
STARKEY, GEORGE W. B.---319 Longwood Ave., Boston 15, Mass.
STEELE, J. D.---Veterans Adm. Hospital, San Fernando, Calif.
STEPHENS, H. BRODIE---384 Post St., San Francisco 8, Calif.
STOREY, CLIFFORD F.---1309 Medical-Dental Bldg., San Diego 1, Calif.
STRANAHAN, ALLEN---Albany Hospital, Albany, N. Y.
STRIEDER, JOHN W.---1180 Beacon St., Brookline 46, Mass.
STRODE, JOSEPH E.---Straub Clinic, Kapiolani St. at Thomas Square, Honolulu 14, Hawaii
SWAN, HENRY, II---4200 East 9th Ave., Denver 20, Colo.
SWEET, RICHARD H.---87 Chestnut Street, Boston 8, Mass.

TABER, RODMAN E.---Henry Ford Hospital, Detroit 2, Mich.
TEMPLETON, JOHN Y., III---1025 Walnut St., Philadelphia 7, Pa.
THOMAS, GORDON W.---Int. Grenfell Association, St. Anthony, Newfoundland
TUTTLE, WILLIAM M.---307 David Whitney Bldg., Detroit 26, Mich.

VAN HAZEL, WILLARD---224 S. Michigan Blvd., Chicago 4, Ill.
VARCO, RICHARD L.---University Hospitals, Minneapolis 14, Minn.
VINEBERG, ARTHUR M.---1390 Sherbrooke St., W., Montreal 25, Que.
VORWALD, ARTHUR J.---College of Medicine, Wayne State Univ., Detroit 7, Mich.

WADDELL, WILLIAM R.---69 Woodland Road, Chestnut Hill, Mass.
WALKER, JAMES H.---1123 Virginia St., E., Charleston 1, W. Va.
WALKUP, HARRY E.---Veterans Adm. Hospital, Oteen, N. C.
WARE, PAUL F.---16 Norwich St., Worcester, Mass.
WATERMAN, DAVID H.---1918 West Clinch Ave., Knoxville 16, Tenn.
WATKINS, ELTON, JR.---Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
WATSON, WILLIAM L.---340 East 72nd St., New York 21, N. Y.
WEBB, WATTS R.---University Hospital, Jackson, Miss.
WEISEL, WILSON---324 E. Wisconsin Ave., Milwaukee 2, Wis.
WHITE, MARION L., JR.---Huntington Bank Bldg., Huntington, W. Va.
WILLAUER, GEORGE---1930 Chestnut St., Philadelphia 3, Pa.

WILLIAMS, MARK H.----63 Front St., Binghamton, N. Y.
WILSON, NORMAN J.----135 Francis St., Boston 15, Mass.
WIPER, THOMAS B.----536 Mason St., San Francisco 2, Calif.
WOODRUFF, WARRINER----8 Church St., Saranac Lake, N. Y.
WOODS, FRANCIS M.----135 Francis St., Boston 15, Mass.
WRIGHT, GEORGE W.----11311 Shaker Blvd., Cleveland 4, Ohio
WYLIE, ROBERT H.----903 Park Ave., New York, N. Y.

## Associate Members

ACKMAN, F. DOUGLAS----1374 Sherbrooke St., W., Montreal 25, Que.
ADELMAN, ARTHUR----751 East 63rd St., Kansas City 10, Mo.
ADKINS, PAUL C.----901 Twenty-third St., N.W., Washington 7, D. C.
AITCHISON, DAVID B.----Mountain Sanatorium, Hamilton, Ont.
ANKENEY, JAY L.----2065 Adelbert Road, Cleveland 6, Ohio

BAFFES, THOMAS G.----Children's Memorial Hospital, Chicago 14, Ill.
BARRETT, RAYMOND J.----18280 Fairfield St., Detroit 21, Mich.
BATTERSBY, JAMES S.----1040 W. Michigan St., Indianapolis 7, Ind.
BENOIT, HECTOR W., JR.----503 Plaza Parkway Bldg., Kansas City 12, Mo.
BERGMANN, MARTIN----4500 Olive St., St. Louis 8, Mo.
BERNATZ, PHILIP E.----Mayo Clinic, Rochester, Minn.
BESKIN, CHARLES A.----3920 Convention St., Baton Rouge, La.
BLAKEMORE, WILLIAM S.----3400 Spruce St., Philadelphia 4, Pa.
BLALOCK, JOHN B.----3503 Prytania St., New Orleans 15, La.
BLOOMBERG, ALLAN E.----1095 Park Ave., New York 28, N. Y.
BLOOMER, WILLIAM E.----333 Cedar St., New Haven 4, Conn.
BOUSQUET, ERNEST O.----5689 Boulevard Rosemont, Montreal, Que.
BROOKS, JAMES W.----1200 E. Broad St., Richmond 19, Va.
BROWN, IVAN W., JR.----Duke University Hospital, Durham, N. C.
BRYANT, JOSEPH R.----321 West Broadway, Louisville 2, Ky.
BURBANK, BENJAMIN----244 Henry St., Brooklyn 2, N. Y.

CAHAN, WILLIAM G.----444 E. 68th St., New York 21, N. Y.
CALLAGHAN, JOHN C.----502 Medical Arts Bldg., Edmonton, Alberta, Canada
CAMPBELL, GILBERT S.----612 N.E. 17th St., Oklahoma City, Okla.
CHANDLER, JOHN H.----The Jackson Clinic, 616 W. Forest Ave., Jackson, Tenn.
CHODOFF, RICHARD J.----255 South 17th St., Philadelphia 3, Pa.
CHUNN, CHARLES F.----442 W. Lafayette St., Tampa 6, Fla.
CINCOTTI, JOHN J.----Veterans Adm. Hospital, Albany, N. Y.
COLE, FRANCIS H.----1375 Goodbar Ave., Memphis, Tenn.
CONNAR, RICHARD G.----706 Franklin St., Tampa 2, Fla.
COOKE, FRANCIS N.----25 S.E. Second Ave., Miami 32, Fla.
COWLEY, R. ADAMS----University Hospital, Baltimore 1, Md.
COX, WILLIAM V.----133 Court St., Auburn, Maine
CRACOVANER, ARTHUR J.----103 East 78th St., New York 21, N. Y.
CRASTNOPOL, PHILIP----1221 East 21st St., Brooklyn 10, N. Y.
CRAWFORD, E. STANLEY----1200 M. D. Anderson Blvd., Houston 25, Texas
CRECCA, ANTHONY D.----376 Roseville Ave., Newark 7, N. J.

DAFOE, COLIN S.----508 Medical Arts Bldg., Edmonton, Alberta
DAMMANN, JOHN F.----632 Preston Place, Charlottesville, Va.

DASCH, FREDERICK W.---416 West Market St., Pottsville, Pa.
DAVILA, JULIO C.---269 S. 19th St., Philadelphia 3, Pa.
DEATON, W. RALPH, JR.---1027 Professional Village, Greensboro, N. C.
DEBORD, ROBERT A.---1240 Jefferson Bldg., Peoria, Ill.
DECKER, ALFRED M., JR.---8 Church St., Saranac Lake, N. Y.
DEMATTEIS, ALBERT---2612 Pleasant Valley Blvd., Altoona, Pa.
DESFORGES, GERARD---49 Lake Ave., Melrose 76, Mass.
DIVELEY, WALTER L.---410 Medical Arts Bldg., Nashville 12, Tenn.
DODDS, G. ALFRED---807 Broadway, Fargo, N. D.

EGLEE, EDWARD P.---115 East 77th St., New York 21, N. Y.

FERGUSON, THOMAS B.---Barnes Hospital, St. Louis 10, Mo.
FINDLAY, CHARLES W., JR.---180 Fort Washington Ave., New York 32, N. Y.
FINNERTY, JAMES---Brookhaven Medical Arts Bldg., Patchogue, N. Y.
FISHBACK, FREDERICK C.---1835 Eye St., N.W., Washington 6, D. C.
FORD, JOSEPH M.---30 East 72nd St., New York 21, N. Y.
FRIEDLANDER, RALPH---The Bronx Hospital, Bronx 56, N. Y.
FRIESEN, STANLEY R.---39th and Rainbow, Kansas City 3, Kan.
FULLER, JOSIAH---205 W. 2nd St., Duluth 2, Minn.

GERBASI, FRANCIS S.---Alexander Blain Hospital & Clinic, Detroit 7, Mich.
GREER, ALLEN E.---1200 North Walker, Oklahoma City, Okla.
GWATHMEY, OWEN---901 23rd St., N.W., Washington 7, D. C.

HAMPTON, FOSTER, JR.---Suite 101, Interstate Bldg., Chattanooga, Tenn.
HANNER, JOSEPH M.---U. S. Naval Hospital, San Diego 34, Calif.
HAUSMANN, PAUL F.---2212 West State St., Milwaukee 3, Wis.
HERBEN, GEORGE F.---House of Rest at Sprain Ridge, Yonkers 2, N. Y.
HEROY, WILLIAM W.---East Gate Road, Lloyd Harbor, Huntington, N. Y.
HERRERA, RODOLFO---11 Calle #2-37, Guatemala City 1, Guatemala
HERTZLER, JACK H.---4377 West Maple Road, Birmingham, Mich.
HEWLETT, THOMAS H.---Brooke Army Hospital, Fort Sam Houston, Texas
HILL, LUCIUS D., III---1118 Ninth Ave., Seattle 1, Wash.
HOOD, R. MAURICE---806 Capital National Bank Bldg., Austin 16, Texas
HUFNAGEL, CHARLES A.---3800 Reservoir Road, N.W., Washington 7, D. C.
HUN, HENRY H.---149 Washington Ave., Albany 10, N. Y.

INGRAM, IVAN N.---655 Sutter St., San Francisco 2, Calif.
IOVINE, VINCENT M.---1150 Connecticut Ave., N.W., Washington 6, D. C.

JAHNKE, EDWARD J., JR.---97th General Hospital, APO 757, New York, N. Y.
JAMPLIS, ROBERT W.---300 Homer Ave., Palo Alto, Calif.
JENSEN, NATHAN K.---1629 Medical Arts Bldg., Minneapolis 2, Minn.
JENSIK, ROBERT J.---224 S. Michigan Ave., Chicago 4, Ill.
JOHNS, THOMAS N. P.---6305 Towana Road, Richmond 13, Va.
JOHNSON, CLIVE R.---1216 Pennsylvania Ave., Fort Worth 4, Texas
JOHNSON, FRANK E.---829 Medical Arts Bldg., Minneapolis 2, Minn.
JOHNSTON, FRANK R.---Bowman Gray School of Medicine, Winston-Salem, N. C.
JUDD, ARCHIBALD R.---304 N. Fourth St., Hamburg, Pa.

KARLSON, KARL E.---451 Clarkson Ave., Brooklyn 3, N. Y.
KAUNITZ, VICTOR H.---4083 Delaware Ave., Tonawanda, N. Y.
KAUSEL, HARVEY W.---Albany Hospital, Albany 8, N. Y.
KAY, JEROME HAROLD---122 North Alta Vista Blvd., Los Angeles 36, Calif.
KEE, JOHN LESTER---3310 Swiss Ave., Dallas, Texas
KEMLER, R. LEONARD---576 Farmington Ave., Hartford 5, Conn.
KENNEY, LEO J.---610 Medical Arts Bldg., Grand Rapids 2, Mich.
KEY, JAMES ALASTAIR---170 St. George St., West, Toronto, Ontario, Canada
KIRSCHNER, PAUL A.---2 East 92nd St., New York 28, N. Y.
KRAEFT, NELSON H.---1501 Magnolia Drive, Tallahassee, Fla.
KUNDERMAN, PHILIP J.---185 Livingston Ave., New Brunswick, N. J.
KUNSTLER, WALTER E.---1538 Sherbrooke St., W., Montreal 25, Que.

LAWRENCE, GEORGE HUGH---1118 Ninth Ave., Seattle 1, Wash.
LEETCH, HENRY W.---108 Main St., Saranac Lake, N. Y.
LEIBOVITZ, MARTIN---812 Medical Arts Bldg., Tulsa 3, Okla.
LEWIS, J. EUGENE, JR.---634 North Grand Blvd., St. Louis 4, Mo.
LEWIS, RUBIN M.---2380 Ellsworth, Berkeley 4, Calif.
LUCIDO, JOSEPH L.---634 North Grand, St. Louis 3, Mo.
LYNN, RALPH B.---R. R. #1, Westbrook, Ontario, Canada
LYON, CLAYTON---384 Post St., San Francisco 8, Calif.

MACDONALD, NEIL---Medical Arts Bldg., Windsor, Ont.
MACPHERSON, LACHLAN---St. John Tuberculosis Hospital, East St. John, N. B.
MADER, VICTOR O.---149 S. Park St., Halifax, Nova Scotia
MADOFF, IRVING M.---1180 Beacon St., Brookline 46, Mass.
MAHONEY, EARLE B.---266 Crittenden Blvd., Rochester 20, N. Y.
MALONEY, JAMES V., JR.---UCLA School of Medicine, Los Angeles 24, Calif.
MANGIARDI, JOSEPH L.---520 Franklin Ave., Garden City, N. Y.
MASON, JAMES M., III---1023 South 20th St., Birmingham 5, Ala.
MCBURNEY, ROBERT P.---899 Madison Ave., Memphis 3, Tenn.
MCGOON, DWIGHT C.---Mayo Clinic, Rochester, Minn.
MELICK, DERMONT W.---1005 Professional Bldg., Phoenix, Ariz.
MENDELSSOHN, EDWIN---1301 Tabor Road, Philadelphia, Pa.
MICHELSON, ELLIOTT---1801 Eutaw Place, Baltimore 17, Md.
MILLER, ARTHUR C.---Loma Linda Medical Center, Loma Linda, Calif.
MILLER, CARROLL C.---304 Humphrey St., Swampscott, Mass.
MORRIS, JOE D.---1801 Weldon Blvd., Ann Arbor, Mich.
MOULDER, PETER V.---950 E. 59th St., Chicago 37, Ill.
MOUSEL, LLOYD H.---Dept. of Anesthesiology, The Swedish Hospital, Seattle 4, Wash.
MUSTARD, WILLIAM T.---200 St. Clair Ave., W., Toronto 7, Ont.

NARDI, GEORGE L.---Massachusetts General Hosp., Boston 14, Mass.
NEALON, THOMAS F.---448 Sabine Ave., Wynnewood, Pa.
NEERKEN, ADRIAN J.---1318 American National Bank Bldg., Kalamazoo 4, Mich.
NELSON, RUSSELL M.---2033 South State St., Salt Lake City 15, Utah
NETTERVILLE, RUSH E.---514 E. Woodrow Wilson Drive, Jackson 6, Miss.
NEWMAN, ROBERT W.---Medical Arts Bldg., Knoxville, Tenn.

O'NEILL, JAMES F.---140 Roslyn Ave., Glenside, Pa.
OVERSTREET, JOHN WM.---508 Hermann Professional Bldg., Houston 25, Texas

PAUL, JOHN S.---Baker VA Center, Martinsburg, W. Va.
PECORA, DAVID V.---Box 20, Ray Brook, N. Y.
PERKINS, REX BEACH---1919 Seventh Ave., South, Birmingham 3, Ala.
PINKHAM, ROLAND D.---Suite 1445, Medical & Dental Bldg., Seattle 1, Wash.
POLK, JOHN W.---604 Medical and Arts Bldg., Springfield 4, Mo.
POTTER, BENJAMIN P.---821 Bergen Ave., Jersey City 6, N. J.
PRATT, LAWRENCE A.---15621 Windmill Pt., Grosse Pointe Pk. 30, Mich.

QUINLAN, JOHN J.---Nova Scotia Sanatorium, Kentville, Nova Scotia

RANSDELL, HERBERT T., JR.---Louisville General Hospital, Louisville 2, Ky.
ROBBINS, S. GWIN---899 Madison Ave., Memphis 3, Tenn.
ROBINSON, JOSEPH L.---1136 West Sixth Street, Los Angeles 17, Calif.
ROSS, RALEIGH R.---2 Medical Arts Square, Austin 5, Texas
RUBENSTEIN, LAURENCE H.---571 Woodlawn Ave., Glencoe, Ill.
RUBIN, MORRIS---2021 Grand Concourse, Bronx 53, N. Y.
RYAN, BERNARD J.---375 East Main St., Bay Shore, N. Y.
RYAN, THOMAS C.---90 Shenango St., Greenville, Pa.

SABISTON, DAVID C.---Johns Hopkins Hospital, Baltimore 5, Md.
SANES, GILMORE M.---3500 Fifth Ave., Pittsburgh 13, Pa.
SELMAN, MORRIS W.---316 Michigan St., Toledo, Ohio
SEYBOLD, WILLIAM D.---Hermann Professional Bldg., Houston 25, Texas
SIRAK, HOWARD D.---Ohio State Univ. Hospital, Columbus 10, Ohio
SKINNER, A. M.---Home Folks Tuberculosis Hospital, Oneonta, N. Y.
SNYDER, HOWARD E.---103½ E. Ninth Ave., Winfield, Kan.
SPENCER, FRANK C.---3500 Tivoly Ave., Baltimore 18, Md.
STATE, DAVID---4833 Fountain Ave., Los Angeles 29, Calif.
STAYMAN, JOSEPH W.---8815 Germantown Ave., Philadelphia 18, Pa.
STENSTROM, JOHN D.---220-1105 Pandora Ave., Victoria, B. C.
STRUG, LAWRENCE H.---2419 Jefferson Ave., New Orleans 15, La.
SULLIVAN, HERBERT J.---Medical Arts Bldg., Hamilton, Ont.
SWENSON, ORVAR---20 Ash Street, Boston 11, Mass.

TAKARO, TIMOTHY---Veterans Administration Hospital, Oteen, N. C.
TAYLOR, FREDERICK H.---1012 Kings Drive, Charlotte, N. C.
TEST, FREDERICK C., II---20252 Meyers Road, Detroit 35, Mich.
TILLOU, DONALD J.---311 W. Church St., Elmira, N. Y.
TIMMES, JOSEPH J.---U. S. Naval Hospital, St. Albans 14, N. Y.
TOCKER, ALFRED M.---209 E. William St., Wichita 2, Kan.
TRICERRI, FERNANDO E.---3 Chemin Mornex, Lausanne, Switzerland

VALLE, A. R.---U.S.P.H.S. Hospital, Detroit 15, Mich.
VAN FLEIT, WILLIAM E.---Emory University Clinic, Atlanta 22, Ga.

WALKER, GEORGE R.---555 University Ave., Toronto, Ont., Canada
WATKINS, DAVID H.---Denver General Hospital, Denver 4, Colo.
WHITESIDE, WILLIAM C.---415 Medical Arts Bldg., Victoria, B. C.
WILKINS, EARLE W., JR.---87 Chestnut St., Boston 8, Mass.
WILSON, JOHN L.---Dept. Surgery, American University of Beirut, Beirut, Lebanon

WITMER, ROBERT H.---126 East Chestnut St., Lancaster, Pa.
WOLCOTT, MARK W.---Veterans Adm. Hospital, Coral Gables, Fla.
WOLFF, WILLIAM I.---30 Central Park South, New York 19, N. Y.

## Senior Members

AMBERSON, J. B.---Bellevue Hospital, New York, N. Y.
AUFSES, ARTHUR H.---812 Park Ave., New York 21, N. Y.

BADGER, THEODORE L.---264 Beacon St., Boston 16, Mass.
BALLON, DAVID---1538 Sherbrooke St., N., Montreal 25, Que.
BARNWELL, JOHN B.---Room 890F, Veterans Adm., Washington 25, D. C.
BAZIN, A. T.---4064 Dorchester St., Westmount, Montreal, Que.
BECK, CLAUDE S.---2065 Adelbert Road, Cleveland 6, Ohio
BENEDICT, EDWARD B.---Massachusetts General Hospital, Boston 14, Mass.
BERRY, FRANK B.---4301 Massachusetts Ave., Washington 16, D. C.
BETTMAN, RALPH B.---104 South Michigan Ave., Chicago, Ill.
BETTS, REEVE H.---Christian Medical College, Vellore, South India
BIRD, CLARENCE E.---64 Alfred Stone Rd., Providence 6, R. I.
BLALOCK, ALFRED---Johns Hopkins Hospital, Baltimore 5, Md.
BLOCH, ROBERT G.---Montefiore Hospital, New York 67, N. Y.
BROWN, A. LINCOLN---490 Post St., San Francisco 2, Calif.
BURNETT, W. EMORY---Broad and Ontario Streets, Philadelphia 40, Pa.
BUTLER, ETHAN FLAGG---956 West Water St., Elmira, N. Y.
BYERS, H. RODDICK---Ganonoque, Ont.

CARLSON, HERBERT A.---4241 East 14th St., Long Beach, Calif.
CARTER, B. NOLAND---Cincinnati General Hospital, Cincinnati 29, Ohio
CHURCHILL, EDWARD D.---Massachusetts General Hospital, Boston 14, Mass.
CLERF, LOUIS H.---5575 Eighth Ave., North, St. Petersburg 2, Fla.
COLE, DEAN B.---Professional Bldg., Richmond, Va.
COOPER, DAVID A.---1520 Spruce St., Philadelphia 2, Pa.
COURNAND, ANDRE---Bellevue Hospital, 27th St. & 1st Ave., New York 16, N. Y.
CURTIS, GEORGE M.---Ohio State Univ. College of Medicine, Columbus, Ohio

DAVIDSON, LOUIS R.---30 East 60th St., New York 22, N. Y.
DECKER, HARRY R.---730 The Park Bldg., 355 5th Ave., Pittsburgh 22, Pa.
DIEFFENBACH, RICHARD H.---570 Mt. Prospect Ave., Newark 4, N. J.
DOLLEY, FRANK S.---2010 Wilshire Blvd., Los Angeles 57, Calif.
DOUGLASS, RICHMOND---Veterans Administration Hospital, Castle Point, N. Y.
DOVELL, CHAUNCEY, COL. (MC), USA (RET.)---62 South Boxwood St., Hampton, Va.

ELOESSER, LEO---490 Post St., San Francisco 2, Calif.

FAULKNER, WILLIAM B., JR.---20 San Rafael Way, San Francisco 27, Calif.
FERGUSON, R. G.---Balfour Apts., Regina, Sask.
FLICK, JOHN B.---1617 Pennsylvania Blvd., Philadelphia 3, Pa.
FREEDLANDER, SAMUEL O.---2460 Fairmount Blvd., Cleveland Heights 6, Ohio

GARLOCK, JOHN H.---47 East 77th Street, New York 21, N. Y.

HARRINGTON, STUART W.---Mayo Clinic, Rochester, Minn.
HARRISON, HARLON W., CAPT. (MC), USNR---U. S. Naval Hosp., San Diego, Calif.
HART, DERYL---Duke University, Durham, N. C.
HAYES, JOHN N.---24 Church St., Saranac Lake, N. Y.
HEAD, JEROME R.---55 E. Washinngton St., Chicago 2, Ill.
HEINBECKER, PETER---Washington University Medical School, St. Louis 10, Mo.
HOLMAN, EMILE---722 Funston Ave., San Francisco, Calif.
HUDSON, WILLIAM A.---602 David Whitney Bldg., Detroit, Mich.

JACKSON, CHEVALIER L.---3401 N. Broad St., Philadelphia 40, Pa.
JANES, ROBERT M.---904 Medical Arts Bldg., Toronto 5, Ont.
JOHNS, FRANK S.---Johnston-Willis Hospital, Richmond 21, Va.
JOHNSON, HOLLIS E.---2122 West End Ave., Nashville 5, Tenn.

KERNAN, JOHN D.---103 East 78th St., New York, N. Y.
KING, DONALD S.---Hitchcock Clinic, Hanover, N. H.
KIPP, HAROLD A.---Mercy Hospital, Pittsburgh 15, Pa.
KNOEPP, LOUIS F.---Veterans Adm. Hospital, Alexandria, La.

LEAHY, LEON J.---105 Medical Arts Bldg., Buffalo 2, N. Y.
LESTER, CHARLES W.---70 E. 80th St., New York 21, N. Y.
LEVEN, N. LOGAN---1464 Lowry Medical Arts Bldg., St. Paul 2, Minn.
LEWALD, LEON T.---1200 Fifth Ave., New York 29, N. Y.
LOCKWOOD, A. L.---300 Bloor St., E., Toronto, Ont.

MEADE, RICHARD H.---Blodgett Medical Bldg., Grand Rapids 6, Mich.
MEYER, HERBERT WILLY---Box 507, Rancho Santa Fe, Calif.
MILLER, ROBERT T., JR.---Mountain Lake, Lake Wales, Fla.
MOERSCH, HERMAN---1064 Plummer Lane, Rochester, Minn.
MOORE, JULIAN A.---208 Doctors Bldg., Asheville, N. C.
MOORE, RICHMOND L. ---180 Ft. Washington Ave., New York 32, N. Y.
MURPHY, JAMES D.---U. S. Veterans Adm. Hospital, Baltimore 18, Md.
MYERS, J. ARTHUR---730 La Salle Bldg., Minneapolis, Minn.

NEUHOF, HAROLD---Box 198, Huntington Road, Stratford, Conn.
NIXON, JAMES W.---1121 Nix Professional Bldg., San Antonio 5, Texas

O'BRIEN, EDWARD J.---307 David Whitney Bldg., Detroit 26, Mich.
OCHSNER, ALTON---Ochsner Clinic, 3503 Prytania St., New Orleans 15, La.
ORNSTEIN, GEORGE---965 Fifth Ave., New York, N. Y.

PACKARD, EDWARD N.---142 Park Ave., Saranac Lake, N. Y.
PICKHARDT, OTTO C.---66 East 79th St., New York, N. Y.
PROCTOR, OSCAR S.---Box 1053, Topeka, Kan.

RAINE, FORRESTER---2230 E. Bradford Ave., Milwaukee 11, Wis.
RIENHOFF, WILLIAM F., JR.---1201 N. Calvert St., Baltimore 2, Md.
RIGLER, LEO G.---Cedars of Lebanon Hospital, Los Angeles 29, Calif.
ROSS, DUDLEY E.---St. Adolphe de Howard, P.Q., Canada

SMITH, DAVID T.---Duke University, Durham, N. C.

THOMPSON, SAMUEL A.----850 Park Ave., New York 21, N. Y.
THORBURN, GRANT----1602 West Genessee St., Flint, Mich.
TOUROFF, ARTHUR S. W.----994 Fifth Ave., New York 28, N. Y.
TYSON, M. DAWSON----Hitchcock Clinic, Hanover, N. H.

VAN ALLEN, CHESTER M.----State Hospital, Bikaner, Rajputana, India

WANGENSTEEN, OWEN H.----University Hospitals, Minneapolis 14, Minn.
WEINBERG, JOSEPH A.----Veterans Adm. Hospital, Long Beach 4, Calif.
WELLES, EDWARD S.----20 Church Street, Saranac Lake, N. Y.
WILSON, JULIUS LANE----Henry Phipps Institute, Philadelphia 47, Pa.

## Members Deceased

DUFF S. ALLEN
DANIEL C. ELKIN
CHEVALIER JACKSON

# ANNUAL MEETING NOTES

The Thirty-ninth Annual Meeting of The American Association for Thoracic Surgery was held in Los Angeles, California, April 21, 22, and 23, 1959. There were 801 physicians who registered for the scientific sessions. In addition there were 87 wives present.

The American Association for Thoracic Surgery has been granted three seats for representatives on the Board of Governors of the American College of Surgeons. These representatives were announced as Drs. George H. Humphreys, II, Edward M. Kent, and Paul C. Samson, serving a one, two, and three year term, respectively. Each year the recurring vacancy will be filled by an Active or Senior member of the AATS who will then serve a regular three year term.

The following changes in the By-laws were approved by the Council and passed by the membership in Executive Session, having been originally proposed by Dr. Richard H. Meade:

Article V, Section 1, of the By-laws should be changed to read as follows:

> The Nominating Committee shall consist of three Active or Senior members who are, by preference, also past Presidents of the Association and in attendance at the meeting. They shall be elected in accordance with the provisions of Article V, Section 1, of the Constitution. The Council shall instruct the Committee as to the vacancies which are to be filled by election.

The following changes in membership status were approved by Council and passed by the membership in Executive Session:

One member was dropped from the rolls of the Association because of nonattendance.

The following Active members were elevated to Senior status:

Arthur H. Aufses
Theodore L. Badger
Edward B. Benedict
Reeve Hawkins Betts
A. Lincoln Brown
W. Emory Burnett
David A. Cooper
Richmond Douglass
Samuel O. Freedlander
Jerome R. Head
Chevalier L. Jackson
Hollis E. Johnson
N. Logan Leven
Oscar S. Proctor
Samuel A. Thompson
Julius Lane Wilson

The following Associate members were elevated to Active membership:

H. William Clatworthy, Jr.
Frederick S. Cross
Preston R. Cutler
George L. Emerson
William W. L. Glenn
Laurence K. Groves
Paul A. Kirschner
Wilford B. Neptune
John M. Snyder
George W. B. Starkey
Rodman E. Taber

The following candidates were elected to Associate membership:

Paul C. Adkins, Washington, D. C.
John B. Blalock, New Orleans, La.
William E. Bloomer, New Haven, Conn.
James W. Brooks, Richmond, Va.
R. Adams Cowley, Baltimore, Md.
Thomas H. Hewlett, Fort Sam Houston, Texas
Lucius D. Hill, III, Seattle, Wash.
Frank E. Johnson, Minneapolis, Minn.
George H. Lawrence, Seattle, Wash.
R. Beverley Lynn, Westbrook, Ontario
James V. Maloney, Los Angeles, Calif.
Dwight C. McGoon, Rochester, Minn.
William T. Mustard, Toronto, Ontario
George L. Nardi, Boston, Mass.
John W. Overstreet, Houston, Texas
David V. Pecora, Ray Brook, N. Y.
Laurence H. Rubenstein, Glencoe, Ill.
Howard D. Sirak, Columbus, Ohio
Joseph J. Timmes, St. Albans, N. Y.
Earle W. Wilkins, Boston, Mass.